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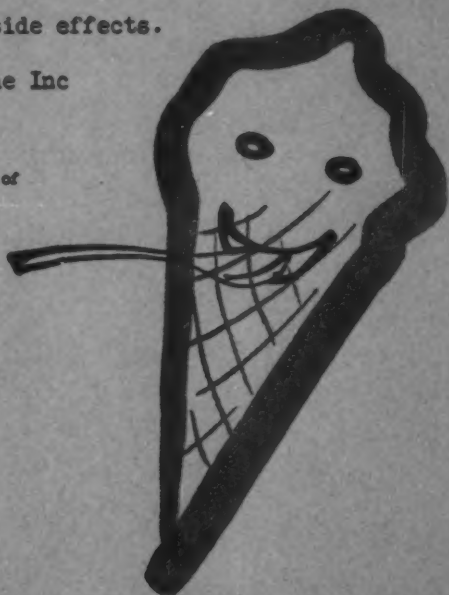
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VOL. LXVI

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No. 1

ACETYLCHOLINESTERASE ACTIVITY IN THE COCHLEA.*

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Since the discovery of "der Vagusstoff" by Loewi¹ and the identification of this material as acetylcholine by Dale², problems in acetylcholine distribution and function have been investigated intensely by physiological and biochemical means. Through recent refined histochemical techniques designed to show discrete localization of acetylcholinesterase³, knowledge of the distribution of this enzyme has been greatly advanced. The number of sites in the nervous system known to contain acetylcholinesterase has been increased almost to the point where this enzyme might be considered to be universally distributed in the nervous system, as Nachmansohn³ contends; yet it is clear that the quantities of acetylcholinesterase differ widely in different neural structures. Acetylcholinesterase was thought to be absent from sensory or afferent neurones⁴ until recently when Koelle⁵ demonstrated that low concentrations of the enzyme do exist at least in several sensory ganglia and nerves.

It is held that the presence of acetylcholinesterase in the nervous system is strongly presumptive evidence that the

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cholinergic mechanism operates where the enzyme is found. Acetylcholinesterase concentrations in any given region of the nervous system have, in general, been found to parallel concentrations of acetylcholine^{6,7} and cholineacetylase⁸, indicating that these three substances occur together. The part played by acetylcholine in the transmission of nerve impulses along axons has not yet been completely elucidated. It is contended by some that acetylcholine is essential for the propagation of nerve impulses, while others hold that the acetylcholine acts only at certain cholinergic nerve endings.⁹

The purpose of the present experiment has been to determine whether or not a cholinergic mechanism is present in the cochlea. If acetylcholinesterase were found in the cochlea, it was hoped that the substance might be localized to particular sites, and that from these data, inferences might be drawn about cochlear function.

Sites of cholinesterase activity were looked for by using the method of Koelle^{10,11,12,13}. This technique, in brief, consisted of incubating tissues in a solution of acetylthiocholine* in the presence of cupric ion. At points of acetylcholinesterase activity, the substrate was hydrolyzed, and the thiocholine liberated formed a precipitate of copper thiocholine. This substance was later converted to copper sulphide when the specimen was treated with ammonium sulphide; thus, copper sulphide, a rather stable and visible precipitate, was seen where acetylcholinesterase had been acting. While the choice of acetylthiocholine for the substrate biased strongly the activity of the cholinesterases in favor of acetylcholinesterase, it was considered wise to verify the type of cholinesterase found by treating some samples with diisopropyl fluorophosphate (D.F.P.)† so that non-specific (pseudo or butyl) cholinesterase would be inactivated; also, for further proof samples were incubated in a solution of butylcholine, some with and some without, D.F.P. treatment.

METHOD.

The experiments were performed on eleven cats and two guinea pigs. The animals were anesthetized by intraperitoneal

* Acetylthiocholine was supplied through the courtesy of LaWall & Harrison Co., 1921 Walnut Street, Philadelphia, Penn.

† D.F.P. was supplied through the courtesy of Dr. Wm. H. Summerson of the Army Chemical Center, Edgewood, Md.

injection of sodium pentobarbital (Nembutal), and were then decapitated, and the cochleae were removed as quickly as possible. The calvarium was opened with a rongeur, the brain extricated, and the petrous bones were removed with large bone-cutting forceps. The cochleae were trimmed of surrounding bone with fine bone-cutting forceps. About one-half of the cochlear wall was removed by fracturing the outer cochlear wall with fine bone-cutting forceps, so that the modiolus and cochlear coils were exposed to view. The scala media was opened throughout a large part of the exposed region, because the spiral ligament separated from its attachment to the basilar membrane and thus was removed with the outer bony wall. Frozen skeletal muscle was sectioned at a thickness of 15 micra and treated with the cochleae. A cochlea and muscle section were treated to demonstrate acetylcholinesterase activity, while another such pair of tissues was treated identically, except that it was not exposed to the substrate. In some experiments such pairs were also incubated in butylthiocholine with and without previous D.F.P. treatment.

Pieces of fractured cochleae were first placed in a storage solution consisting of 9.0 ml. of 40 per cent sodium sulfate and 6.0 ml. water, kept at a temperature of 33°-35° C. As soon as possible the tissues were transferred to Coplin jars, which contained the incubating solutions in which the tissues were kept for 45 minutes at 38° C.

The acetylcholinesterase incubating solution consisted of 9.0 ml 40 per cent sodium sulfate, 0.6 ml. copper glycine, 1.5 ml. sodium acid maleate, 0.6 ml. magnesium chloride, and 2.1 ml. water. To this solution, prepared 15 minutes before the time of use, was added 1.2 ml. of acetylthiocholine solution. This was prepared by placing 23 mg. of acetylthiocholine iodide in a centrifuge tube with 1.2 ml. water and 0.4 ml. of 0.1 M copper sulfate. After centrifuging the copper iodide down, the supernatant fluid was decanted into the incubating solution, which was then filtered just prior to use; also, before the tissues were transferred to the incubating solution a trace of copper thiocholine was added.

Control tissues were placed in a solution containing 6.0 ml.

of 40 per cent sodium sulfate, 0.4 ml. copper glycine, 1.0 ml. sodium acid maleate, .04 ml. magnesium chloride, 1.4 ml. water and a trace of copper thiocholine.

At the end of the incubating period, all tissue samples were placed in 10.0 ml. 40 per cent sodium sulfate and 10 ml. water with a trace of copper thiocholine for five minutes. They were then transferred to 5 ml. 40 per cent sodium sulfate and 16 ml. water with copper thiocholine for one minute. They were next passed through water containing a trace of copper thiocholine for one minute. After this, the tissues were developed in a solution of 1.0 ml. saturated ammonium sulfide solution diluted with 30 ml. water which had been saturated with copper sulfide. The specimens were then rinsed in water for one second and were placed in 10 per cent formalin saturated with copper sulfide. The tissues which were treated to exclude non-specific cholinesterase were first of all placed in 1.5 ml. of one millionth mole D.F.P. with 4.5 ml. water and 9.0 ml. 40 per cent sodium sulfate for 30 minutes at 35° C. Frozen sections of striated muscle, 10-15 microns thick, were placed on slides, allowed to dry one minute, and were then run through the same process.

Those specimens which were treated with the substrate butylthiocholine were divided into two groups as to whether or not treated with D.F.P. Those that were not treated with D.F.P., and thence were run to show non-specific cholinesterase, were first placed in a storage solution composed of 10.5 ml. 40 per cent sodium sulfate and 4.5 ml. water. The incubation solution for these tissues was composed of .6 ml. copper glycine, 1.5 ml. sodium acid maleate, .6 ml. magnesium chloride, 10.5 ml. sodium sulfate, .6 ml. water, 1.2 ml. butylthiocholine, and a trace of copper thiocholine. D.F.P. treated specimens used as controls were placed in an incubation solution containing .4 ml. copper glycine, 1.0 ml. sodium acid maleate, 6.4 ml. magnesium chloride, 6.0 ml. sodium sulfate, 1.4 ml. water, .8 ml. butylthiocholine and a trace of copper thiocholine. The butylthiocholine was prepared by placing 43 mgms. butylthiocholine iodide, 1.8 ml. water, and .6 ml. of .1 molar copper sulfate in a centrifuge tube, the filtrate being saved and used.

Reagents were composed as follows:

Copper glycine: 3.75 gm. glycine, 2.50 gm. $\text{CuSO}_4 \cdot 5\text{H}_2\text{O}$, q.s. 100 ml.

Maleate Solution: 9.60 gm. NaH maleate, 52.2 ml. N NaOH, q.s. 100 ml.

Sodium Sulfate: 40 per cent weight per volume Na_2SO_4 , adjusted to pH 6.00 at 38° C.

Magnesium Chloride: 9.52 gm. MgCl_2 , q.s. 100 ml.

Copper thiocholine was obtained by filtering the incubation solution after use and storing for two to four days. The precipitate was collected by filtration.

After fixation, the cochleae were placed in a shallow bath of Ringer's solution containing copper sulfide. The tectorial membrane was removed under the dissecting microscope, and pieces of the organ of Corti and osseous spiral lamina were teased from the cochlea and mounted on slides in Apathy's gum arabic. Some cochleae were counter-stained, embedded in paraffin and sectioned; others were gold-toned, decalcified, embedded in celloidin and sectioned.

RESULTS.

All acetylthiocholine treated cochleae contained a dark stripe lying in the region of the organ of Corti, which was seen clearly with the naked eye (see Fig. 1). In no experiment did the controls have this precipitate on gross and microscopic examination. When examined under the dissecting microscope it was seen that the dark stripe faded away as the cochlear duct entered the unopened regions of the cochlea. Under high magnification the stripe in most cochleae was resolved into two parallel bands, the inner band being much more intense than the outer band (see Fig. 2). The precipitate was seen in all turns of these cochleae, but no attempt was made to study the relative density of the precipitate in the several turns.

While variable accessibility of chemical solutions to different cochlear regions would seem to be important in evaluating the relative densities of precipitate in the different cochlear turns, no such factor should play a role in the comparison of precipitate density of inner and outer bands, particularly

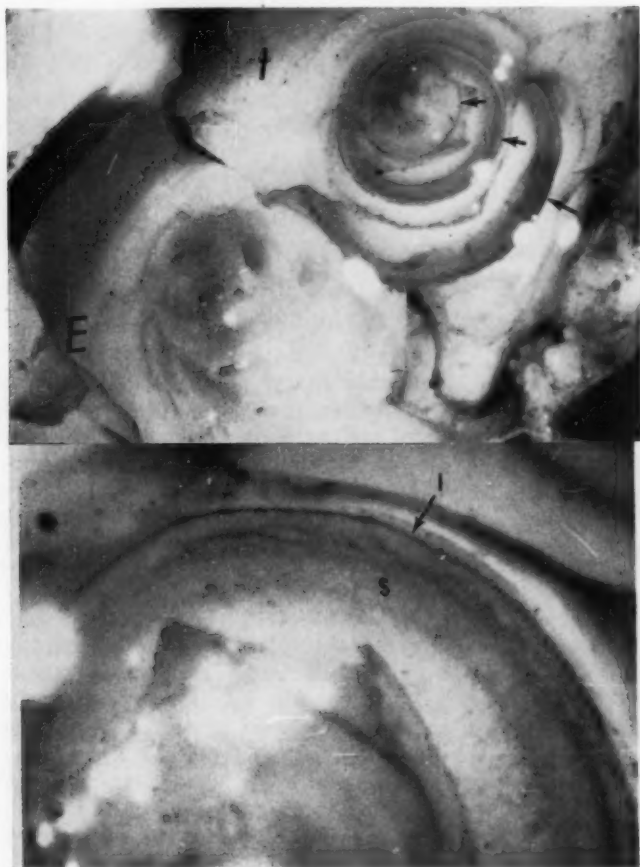


Fig. 1.

when the outer band, which is the least dense, has the most direct exposure to the solutions.

Under the higher power of the dissecting microscope, the inner band was observed to lie near the outer edge of the limbus in the inner hair cell region; and the outer band, much

broader than the inner, was seen to consist of three lines lying in the region of the three rows of outer hair cells.

Examination of the teased specimens mounted on slides and the microscopic sections confirmed the impression that the precipitate lay very close to the hair cells (see Fig. 3). The inner band of precipitate, although restricted to an area with



Fig. 2.

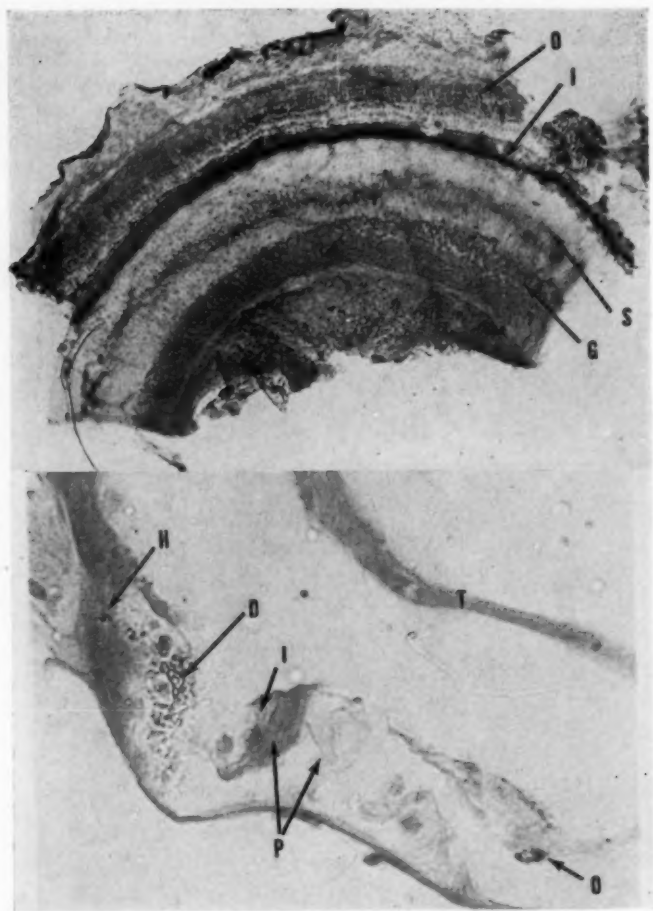


Fig. 3.

discrete boundaries, was so broad and dense that adjacent cytological detail was largely obscured. The outer band consisted of small patches of precipitate arranged in rows. These patches were ovate to cupulate in form, measured 2-5 micra in diameter, and lay in close relationship to the rows of outer

hair cells. The precipitate was limited to areas consistent in position and size with the chalices of the hair cells. A dense amorphous mass was seen lying in the region between the habenula perforata and the inner hair cells, extending beyond the confines of the hair cell chalices or nerve end-

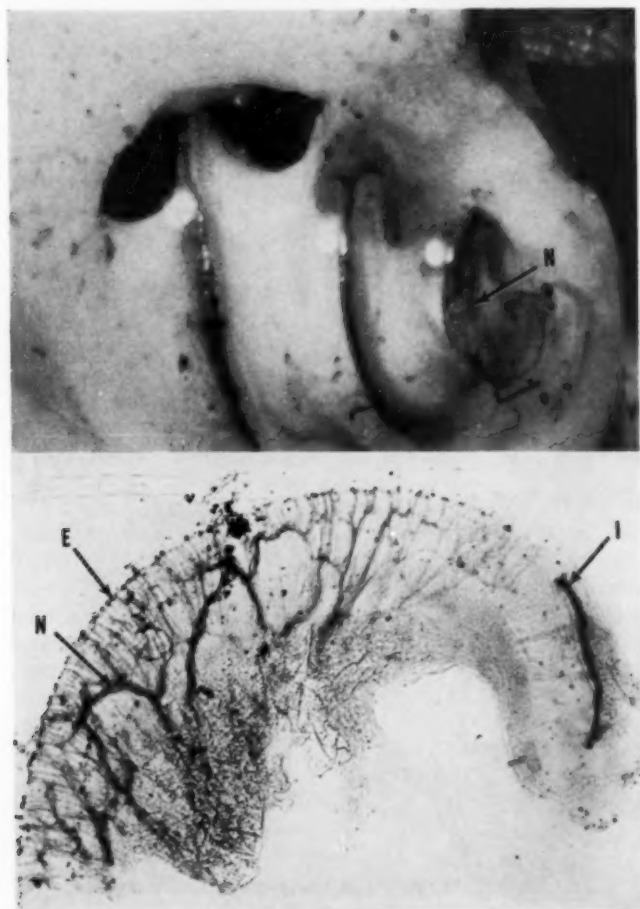


Fig. 4.

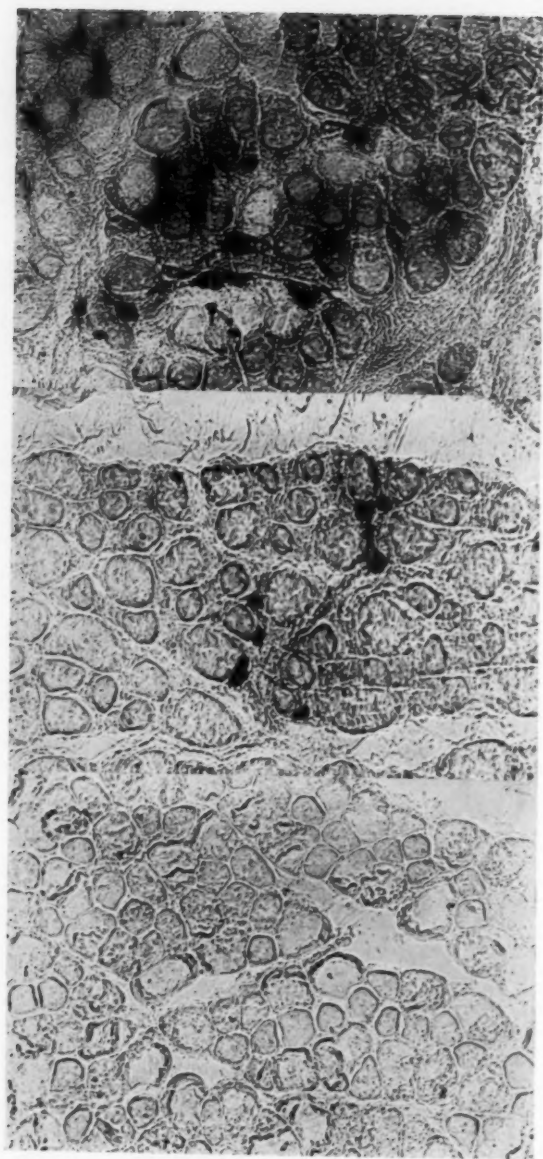


Fig. 5.

ings. In some areas fine lines of precipitate appeared to outline individual nerve fibers (see Fig. 4). These outlined fibres were most numerous near the habenula perforata, becoming sparser near the spiral ganglion. Since deeper parts of the specimen, such as the spiral ganglion, were not exposed adequately to the chemical solutions, the experiment does not provide information about these structures.

The muscle sections provided a check on each experiment to prove that the proper technique had been followed. In the muscle treated with acetylthiocholine the motor-end plates contained a dense precipitate, whereas in the muscle not treated with the substrate, the motor-end plates contained no precipitate (see Fig. 5). The density of precipitate bands in the organ of Corti and of the motor-end plates was not altered by incubation in D.F.P.; however, surrounding tissues were paler than non-D.F.P. treated specimens. This suggests that the slightly brownish discoloration of the specimens not treated with D.F.P. was due to the action of non-specific cholinesterase and thioesterases.

DISCUSSION.

The heavy brown amorphous precipitate found in the organ of Corti was believed to be copper sulfide found at sites where acetylcholinesterase had been hydrolyzed by acetylthiocholine. That these deposits were the results of acetylcholinesterase activity rather than from activity of non-specific cholinesterases or thioesterases, was borne out by the results of treatment of control specimens with butylthiocholine and D.F.P. Occasionally needle shaped crystals were seen in locations of high acetylcholinesterase activity. These were thought to be copper thiocholine¹⁴ which had escaped complete conversion to copper sulfide, indicating that in these regions acetylthiocholine had been hydrolyzed by acetylcholinesterase. It was concluded that acetylcholinesterase was present in the organ of Corti intimately associated with the hair cells and their nerve endings.

According to the findings presented here, acetylcholinesterase found in the organ of Corti may be related to the hair cells, endings of the afferent neurones, or endings of the efferent fibres from Rasmussen's bundle. Derbyshire and

Davis,¹⁵ in 1935, considered the possibility of a chemical mediator in the conversion of sound energy to acoustic nerve action potentials. They calculated that the latency of 0.5 to 0.7 msec. between the microphonic cochlear response and onset of the spike of the action potential was of an order comparable with the rates of release, action and destruction of acetylcholine.

Also neural summation for high frequencies might be explained by a chemical transmission hypothesis. For frequencies of 2000 cps and lower each volley of nerve impulses, as revealed by the action potential, can be clearly associated with one sound wave or another, but when the frequency is higher, *e.g.* 8000 cps, the nerve responds to a tone pip as a whole instead of to individual waves¹⁶; thus, the stimulating effect seems to be integrated by some process, termed "summation," in which the nerve responds as a whole instead of to the individual waves. The explanation might be that a chemical or enzyme reaction occurring at the sensory-neural junction is so rapid that for low frequencies the chemicals are deposited and destroyed before the onset of the next stimulating wave. The decay time is too slow, however, for it to be completely destroyed in the time interval between waves of a high frequency pip, so that a build-up would occur until adequate to trigger the action potential.

Gisselsson¹⁷ recently made a searching review of the chemical mediator theory and confirmed the finding of Martini^{18,19} that there were cholinesterases in these fluids. He obtained cat perilymph and endolymph by pipetting the fluids out through punctured round windows and basilar membranes. He then incubated measured volumes of the fluids with acetylcholine of known concentration, and compared contractions of leech dorsal muscle and frog rectus muscle bathed in the solutions with contractions of the indicator muscle in acetylcholine of known concentration. By this procedure values for cholinesterase activity were obtained.

Although acetylcholinesterase has been found in sensory neurones by Koelle⁵, the concentrations were evidently quite low. The presence of such a conspicuous amount of the enzyme in the organ of Corti suggests that it may be related to the

efferent nerve endings rather than to the hair cells of acoustic nerve endings. The efferent fibers were first described by Rasmussen^{20,21} and called by him the "olivo-cochlear tract." The cells of origin of this tract lie medial to the superior olivary nucleus and dorsal to the nucleus of the trapezoid body in the medulla. The tract decussates hard by the colliculi of the facial nerve and passes into the inferior division of the vestibular nerve and then by three fascicles in a spiral course up the cochlea. The termination points of these fibers have not been discovered, although experiments of Portmann²² and of Schuknecht²³ suggest that they end upon the inner hair cells. At any rate, it is quite certain that these fibers do not end upon blood vessels, or structures apart from the organ of Corti.

Experiments by Galambos²⁴ where amplitudes of action potentials of the acoustic nerve in response to clicks were measured while the olivo-cochlear bundle was being stimulated, revealed that the stimulated tract produced an inhibitory effect upon acoustic nerve potentials. It is considered possible that the olivo-cochlear bundle through a cholinergic mechanism exerts an inhibitory or facilitory effect upon the hair cell acoustic nerve junction, depending upon the amount of acetylcholine released. Experiments are now in progress which, it is hoped, will clarify this matter.

SUMMARY.

1. Acetylcholinesterase was found in the organ of Corti of the cat.
2. The enzyme appeared to be within or near the nerve chalice for the hair cells rather than upon or in the hair cells themselves.
3. The row of inner hair cells showed a much greater indication of acetylcholinesterase activity than the outer hair cell rows.
4. Many nerve fibers oriented toward the organ of Corti also showed strong acetylcholinesterase activity.

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THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY CONVENTION CRUISE

The Virginia Society of Ophthalmology and Otolaryngology is sponsoring a convention cruise to Havana and Nassau on May 26 to June 2, 1956. Sailing from and returning to Norfolk, Virginia, the "Queen of Bermuda" will act as the hotel for the trip. Fare for seven days, \$165.00 and up per person. Make reservations with United States Travel Agency, Inc., Washington, D. C.

THE EAR IN HEAD TRAUMA.*†

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The temporal bone is frequently involved in craniocerebral injury. Since it houses the cochlear and vestibular mechanisms, as well as the VIIIth cranial nerve, injuries in this neighborhood may cause serious sequelae. The object of this report is to present the mechanics involved in skull fractures and to review the otological disturbances resulting from head injuries. The subject is worthy of scrutiny because of the many new concepts which have been formed as a result of recent experimental and clinical studies on many of the problems bearing on the subject.

CLASSIFICATION AND MECHANISM OF SKULL FRACTURE.

One can usually predict in a general way the type of fracture that will result with a given intensity of force applied to a specific region of the skull. Variations are dependent upon the contour and consistency of the skull. This was determined by stresscoat and strain gauge studies.^{1,2} This discussion will concern fractures involving the temporal bone.

The petrous portion of the temporal bone forms two-thirds of the floor of the middle fossa and one-third of the floor of the posterior fossa and, therefore is vulnerable in fractures involving the base of the skull. The foramina of the petrosa act as areas of stress concentration. Consequently, fracture lines in the vicinity may extend toward these foramina.

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The types of blows commonly associated with fractures of the temporal bone may be classified as follows:

1. Temporal and parietal area blows.
2. Occipital area blows.
3. Frontal area blows.

TEMPORAL AND PARIETAL AREA BLOWS.

These result in tearing apart forces of the base in an anteroposterior direction. A fracture produced by such a blow is vertically directed in the parietal and temporal bones, and may extend toward the base of the skull and top of the petrosa. Even though the petrosa is a buttress and may direct a fracture line anteriorly between it and the spheno-zygomatic buttress, yet when the stress is at right angles to the petrosa a fracture will frequently split or bisect it lengthwise, producing the commonly called longitudinal fracture. In severe cases the petrosa may become comminuted and fragmented with the fracture line continuing on into the foramen magnum (see Fig 10).

TYPES OF PARIETAL BLOWS:

1. Posteriorly to one side of the midline; frequently involves the bony external auditory canal (see Fig. 11).
2. Posterior edge of interparietal area at midline; (see Fig. 12). The fracture often extends into the mastoid process
3. Low, anterior parietal area; fracture line extends down and forward toward the anterior portion of the temporal fossa to involve the temporal squama and the greater wing of the sphenoid. The fracture may extend to the body of the sphenoid and even across to the other side (see Fig. 7).

Longitudinal fractures of the petrosa are usually extensions from fractures of the temporoparietal region passing down toward the external auditory meatus and then along the anterior aspect of the petrosa toward the Gasserian fossa.

OCCIPITAL AREA BLOWS.

These result in tearing apart forces of the base from side to side, particularly in the occipital squama (posterior fossa).

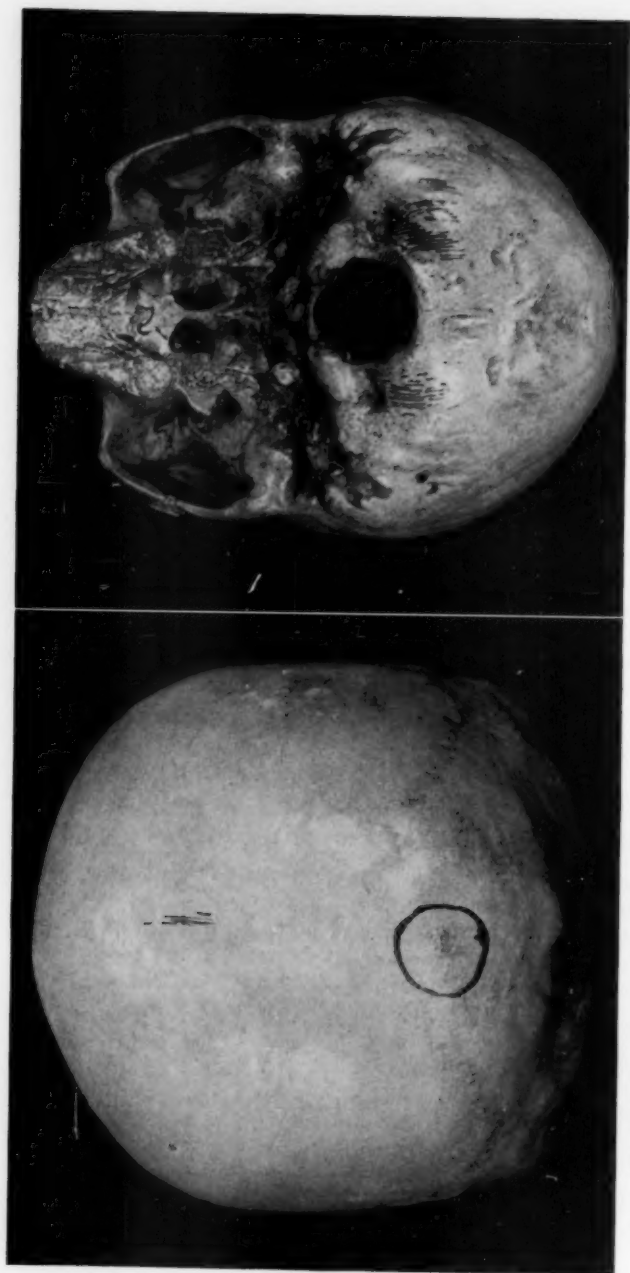


Fig. 1. Stresscoat patterns following a mid-occipital blow in a cadaver's skull. Note that at the base of the skull there are tearing apart forces from side to side about the foramen magnum and the squamous portion of the occipital. Note also the tearing apart forces shown on the condyloid process of the occipital. In the view above there is evidence of minor tearing apart forces in the interparietal region. The area of impact is shown by the black circle. The blow in this location can conceivably cause fracture to extend through the petrous bone and bisection it on one or both sides transversely.

The fracture begins either in the vicinity of the foramen magnum or more laterally to involve the hypoglossal and jugular foramina. If the energy is adequate, the fracture extends forward through the basilar process of the sphenoid, fractures the petrosa transversely and ends in the middle fossa (see Figs. 1, 2, 3).

Transverse fractures of the petrosa are almost always

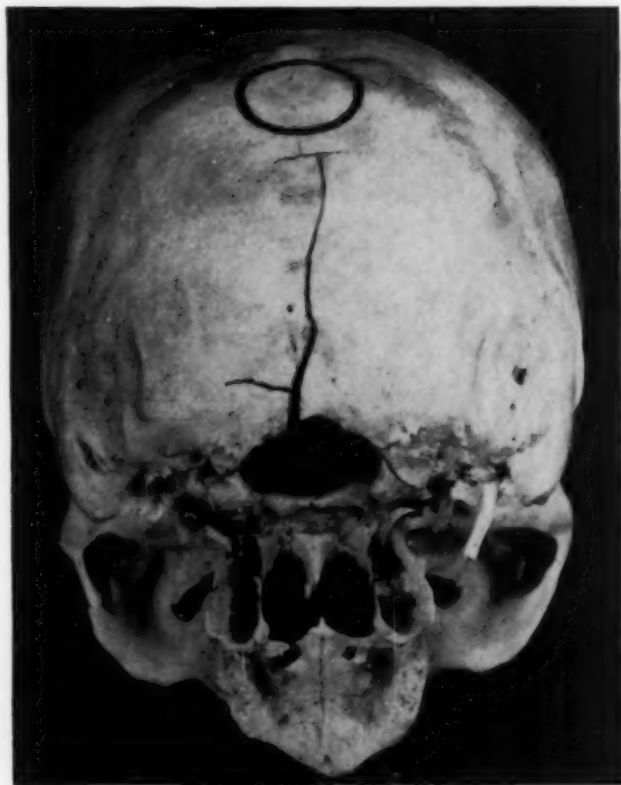


Fig. 2. Experimental skull fracture. Area of impact shown by black circle. The fracture line extending into the foramen magnum is much wider at the foramen than it is at the point of impact. Extensions of fracture toward the jugular foramen on both sides are noted. They also involve the condyloid process of the occipital.

extensions of posterior fossa fractures. They may, however, occasionally result from a posterior extension of an anterior fossa fracture (see Fig. 4).

In transverse fractures there is usually involvement of the internal acoustic meatus with severe injury to the VIIIth cranial nerve, resulting in a loss of cochleo-vestibular function. The VIIth nerve is often transected.



Fig. 3. Experimental fracture due to a blow in the midline occipital area resulting in a transverse fracture of the petrous bone on the right side as well as a fracture of the basilar process of the occipital.

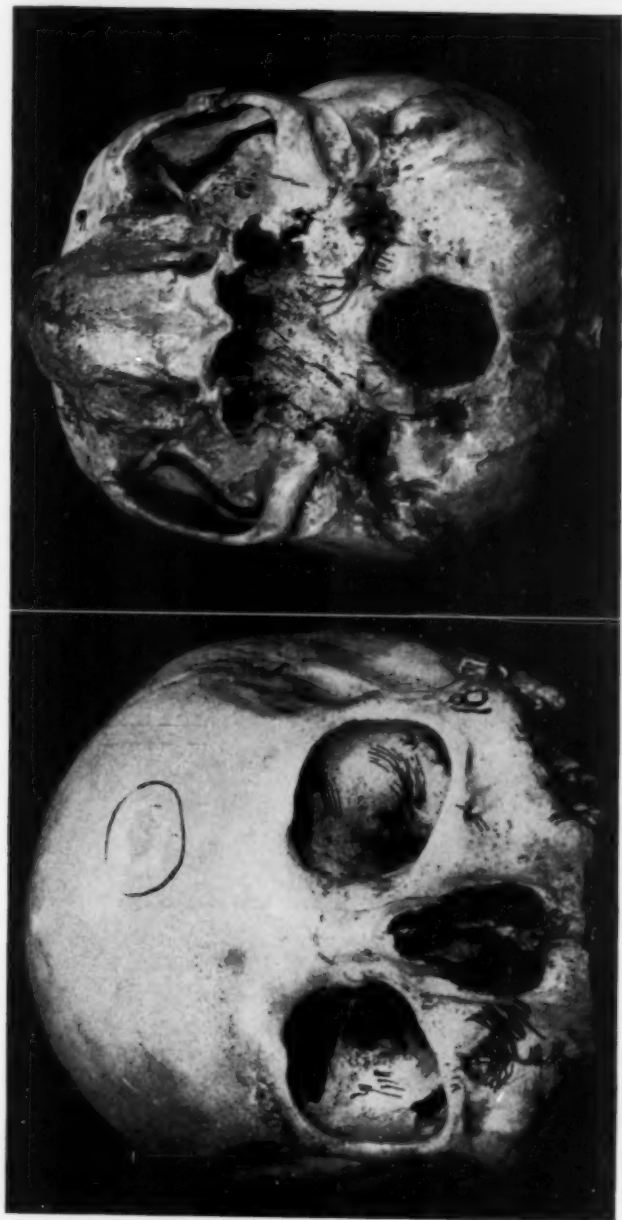


Fig. 4. A left frontal blow in a cadaver's skull showing tearing apart forces of the orbita and the upper jaw as well as the base of the skull. Note that with a blow of sufficient severity a fracture line may extend across the base of the orbita and the upper jaw from the left to the right side. Such a fracture may involve the petrous bone on the same or on the opposite side and in an oblique-transverse direction.



Fig. 5. Autopsy findings associated with a depression in the left fronto-temporal area with a fracture extending to the base of the anterior fossa, to the pituitary region and then across the length of the petrous bone on the opposite side.

Pure transverse or longitudinal fractures are uncommon. In either type comminution and fragmentation is usually present; furthermore, modifications and combinations of transverse and longitudinal fractures of the petrosa may occur. Combined longitudinal and transverse fractures may occur from severe blows upon the head, particularly in falls upon the vault from

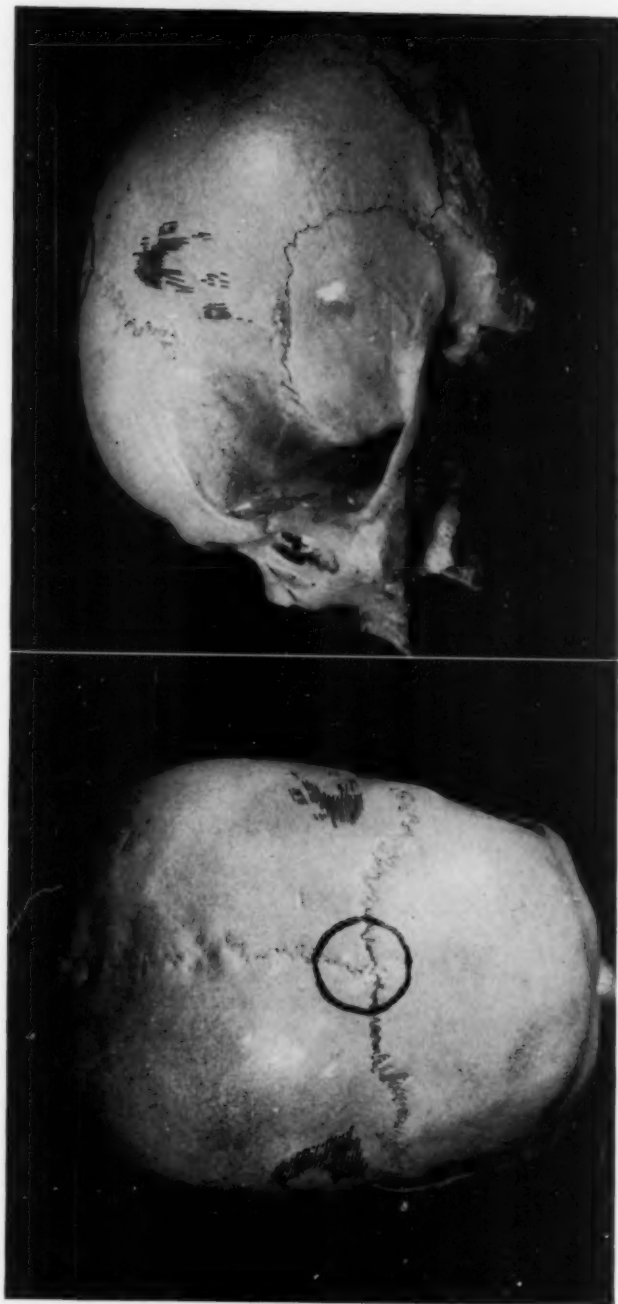


Fig. 6. An interparietal blow at the frontal junction showing tearing apart forces involving the parietal bones on both sides. The tearing apart forces are in an anteroposterior direction with the cracks in the lacquer extending superoinferiorly. Such a blow may conceivably cause unilateral or bilateral fracture extending toward the mastoid and toward the external auditory canal. Such a fracture can cause a longitudinal involvement of the petrous bone.



Fig. 7. A temporo-parietal blow resulting in tearing apart forces antero-posteriorly of the skull. Such an injury may result in a longitudinal fracture of the petrous bone on the one or both sides.

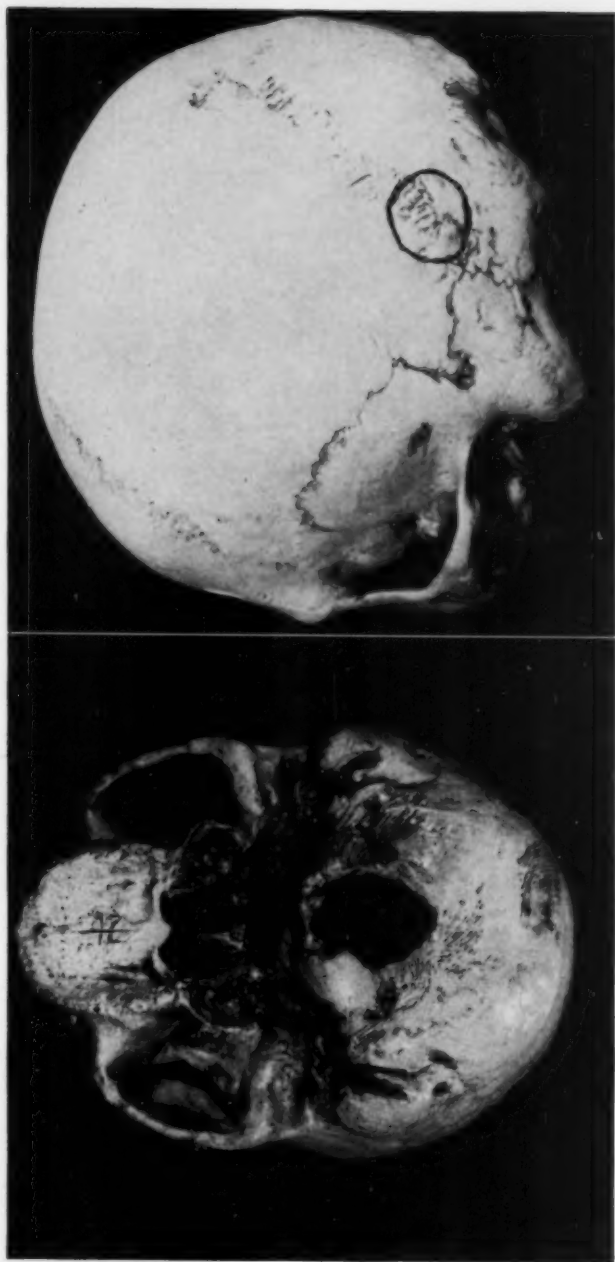


Fig. 8. Impact above mastoid on left resulting in tearing apart forces on base slanting obliquely toward both petrous bones and the foramen magnum.



Fig. 9. Experimental fracture following a posterior vertex blow with lines of fracture extending to the foramen magnum in the midline posteriorly and toward the petrous bone, from the parietal down to the temporo-mastoid area. The fracture line involves the petrous bone longitudinally as seen in this figure. The petrous involvement is obliquely forward and medially.

a good height. Figure 10 illustrates a vault fracture extending toward the petrosa. It was caused by falling three floors and landing on the head. At autopsy the fracture was found to extend through the petrosa transversely and thence through the jugular and hypoglossal foramina to the foramen magnum, so that a triangular piece of bone was actually broken loose. The right XIIth nerve was paralyzed.



FIG. 10. A parietal fracture extending down toward the mastoid area has involved the petrous bone longitudinally and the occipital bone surrounding the foramen magnum. This patient had a twelfth nerve involvement before death. Fracture extensions involving the condyloid foramen were noted at autopsy.

FRONTAL BLOWS.

These result in tearing apart forces from side to side at the base of the skull in the anterior fossa. A frontal fracture involving the anterior fossa may extend into the middle fossa,

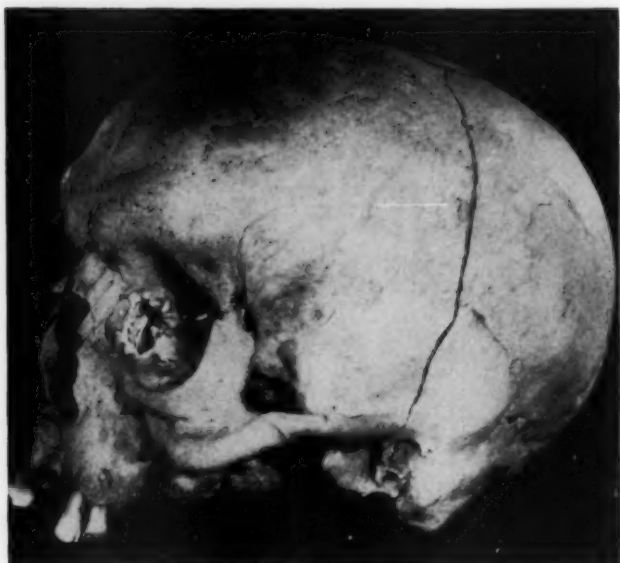


Fig. 11. Experimental skull fracture with impact at the vertex (circle). Note the involvement of the external auditory canal. Inside the skull such a fracture involves the length of the petrous pyramid ending in the region of the lateral pituitary area.

fracture the petrosa transversely and extend on into the posterior fossa (see Fig. 4). A lateral frontal blow (see Fig. 5), may result in an obliquely directed fracture of the base of the skull.

ISOLATED FRACTURES.

Isolated fractures of the base, unassociated with radiations into the vault, may involve the middle fossa and petrosa, but these are not common. In posterior parietal, interparietal and mid-occipital blows, such isolated stresses of the base were seen in about six per cent of the skulls studied. These fractures may be produced by:

1. Blows upon the vault.
2. Blows so applied as to cause an inbending about the foramen magnum from compression of the head against the neck or vice versa.

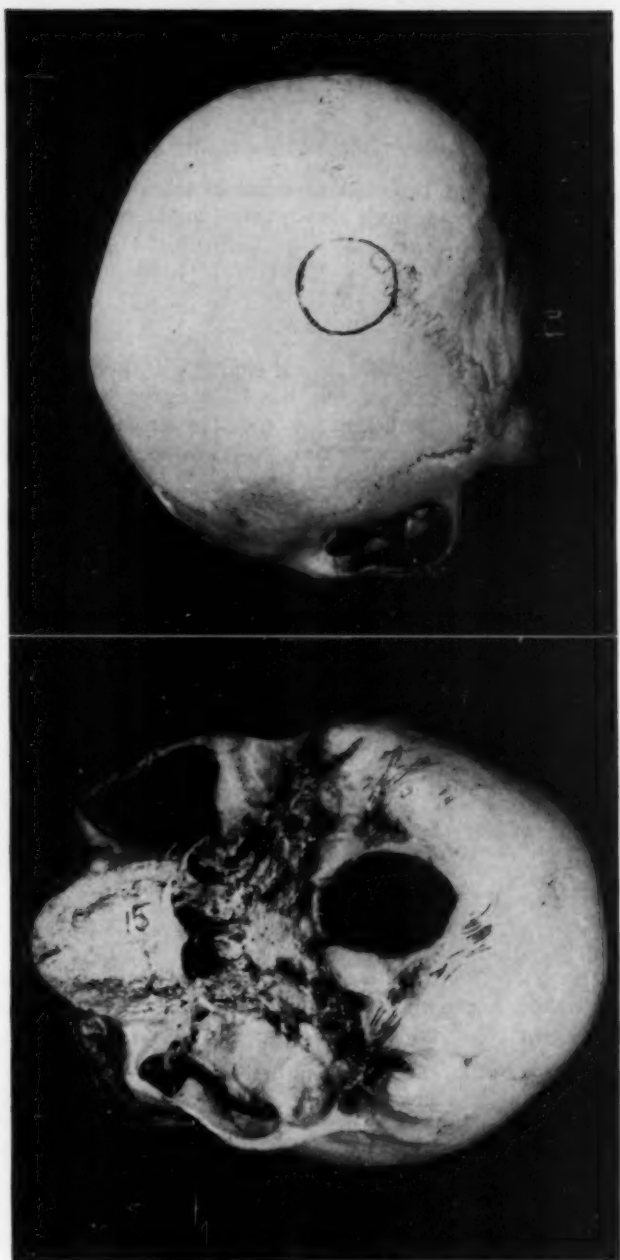


Fig. 12. A left lateral parieto-occipital blow near the mastoid resulting in tearing apart forces obliquely across the base of the skull in the occipital area extending toward the foramen magnum and toward the petrous bone on the opposite side.

3. Blows applied to the temporo-mandibular joint.
4. Blows low along the circumference of the skull, particularly by sharp objects, may also involve the base primarily very much like a hatchet may tear through the grain of a piece of wood.⁶⁴

PATHOLOGY.

Our knowledge of the pathological changes in the temporal bone as a result of head trauma, has been derived from autopsy studies (gross and microscopic examination of the temporal bone) and from animal experiments reported by various investigators.

The pathology in longitudinal fractures:^{3,4,5}

1. The fracture courses through the roof of the middle ear and then along the anterior edge of the pyramid (through the superior wall of the Eustachian tube and carotid canal).
2. The tympanic membrane and external canal walls were often injured. The incus may be dislocated and the ossicular ligaments torn. The ossicle may be fractured.⁴
3. The labyrinthine capsule remains undamaged.
4. If the facial canal is involved, the damage usually occurs in the region of the genu. The facial nerve is usually spared.
5. The nerves to the internal ear are almost always injured by hemorrhage, tearing or stretching.
6. The vestibular end-organ is rarely damaged.
7. There is frequent evidence of injury to the membranous cochlea (hemorrhages into spiral ligament and scala tympani) especially near the round window.
8. Deafness is more of the conduction type. Occasionally inner ear deafness is pronounced and may be due to isolated cochlear fractures, direct damage to the hair cells by the traveling pressure wave, or damage to the higher centers.

The pathology in transverse fractures:³

1. The fracture courses at right angles to the pyramid.

2. The cochlea is more vulnerable than the vestibule.
3. The nerves in the internal auditory meatus may be torn.
4. There may occur a tearing of the membranous labyrinth.
5. Hemorrhages occur into endolymph as well as into perilymph.
6. The middle ear damage, if present, is confined to the mesial wall (musculo-tubal canal, knee of the facial, stapes footplate).
7. The facial nerve is often involved especially at the geniculate ganglion (in over 50 per cent of the cases). The paralysis may be permanent.
8. Hematotympanum occurs at times. External bleeding from the ear is rare.
9. Cerebrospinal otorrhea is more common in transverse than in longitudinal fracture. This fluid may drain down the Eustachian tube.

The pathology of isolated labyrinthine fractures has been reviewed by Grove.⁶ Usually if the labyrinthine capsule is fractured, a complete loss of cochlea-vestibular function ensues. Occasionally, however, an isolated fracture of either cochlea or vestibule may occur with an isolated loss of function of one portion and a partial to complete preservation of function of the other portion of the labyrinth.

Studies of inner ear changes after mild injuries were made in animals by Stenger.⁷ Following light blows with a hammer on the skulls of white rats, he found hemorrhages in the region of the round windows and in the basal coil of the cochlea. The vestibule showed no changes. With heavier blows the hemorrhages were more severe at the round window and extended to the helicotrema. Some bleeding was noted in the cochlear nerve and in the ampullae. Similar experiments were conducted by Brunner⁸ on guinea pigs, and they confirmed Stenger's findings.

Pathological changes found in the temporal bones of those dying soon after injury: the changes found microscopically in animal experiments and the changes found in the temporal

bones of those who died many years after head trauma, are quite similar. Grove⁶ states that these changes consist of stretching and tearing of nerves to various degrees, hemorrhages in various locations but particularly in the perilymphatic spaces of the basal coil of the cochlea in the vicinity of the round window. "The exudation of blood is probably partly absorbed and partly organized, leading to atrophy of the finer nerve branches and degeneration of the neuroepithelium of both cochlea and vestibule."

In the event of extensive bleeding into the labyrinth, there is a strong likelihood of extensive connective tissue organization of the blood clots. Later, this reparative connective tissue invasion may in turn be replaced by new bone formation within the confines of the osseous labyrinth.^{3,9}

Labyrinthine fractures heal with new bone formation in the endosteal and periosteal layers. The enchondral layer heals only with connective tissue.¹⁰ The healing is fairly well advanced in one month. Occasionally these fractures appear to heal incompletely or not at all, so that subsequent middle ear infection might well diffuse through these old fracture lines and reach the meninges. These unhealed fractures may be discernible in Roentgen studies many years later.

Many cases of head trauma, without clinical or radiological evidence of skull fracture, develop deafness and vestibular disturbances. This may be due to hemorrhages into the labyrinth^{4,7,8,11} or to severe degenerative changes in Corti's organ^{12,13,14} or to pulling and tearing of the cochlear nerve in the internal auditory meatus.^{3,15,16}

The term "commotio labyrinthi" has been used to describe the presence of a hemorrhage into the inner ear. Grove⁶ considered labyrinthine concussion or commotio as the commoner cause of deafness and vestibular disturbances. He pointed out that hearing loss with a fractured skull and contralateral hearing loss in fracture cases can be explained on the basis of a commotio or inner ear hemorrhage. Ballance¹⁷ has used the term "concussion" to describe this state. Ballance believed that hearing loss may result not only from injury to the peripheral mechanisms but also from damage to the central auditory pathways by the commotion.

Oppenheim suggested that *commotio cerebri* may lead to hearing loss by injury to the cochlear nuclei or higher centers. Because of the partial crossing of the ascending acoustic fiber systems, central lesions will give few if any symptoms²⁷. Lesions of the medial geniculate body or the auditory cortex will not result in deafness unless bilateral, according to Tobey.¹⁸

Normal auditory thresholds in the cat were found by Neff after bilateral destruction of the auditory cortex.¹⁹ Schuknecht and Woellner have shown that up to 75 per cent of nerve fibers supplying a particular region of the cochlea can be destroyed without creating threshold losses for frequencies having their locus in that region.^{20,21} The clinico-pathological evidence of true central deafness is inconclusive. Fraser and Rowe²² reported a reduction in hearing in some instances of temporal lobe tumors. Unilateral removal of the temporal lobe has not influenced hearing, according to Penfield and Erickson.²³

Moderate blows with a hammer were delivered to the exposed mastoid of human volunteers by Ruedi and Furrer²⁴ and by Escher.^{25,26} The subjects described an acoustic sensation resembling a pistol shot. Audiograms showed mild 4,000 cycle dips which disappeared after 24 to 48 hours. Lindsay stated that the blows of a mallet at mastoidectomy may cause permanent hearing loss at 4,000 cycles.²⁷

Schuknecht, Neff, Perlman²⁸ subjected conditioned cats to head blows. Audiograms were taken before and after the trauma. They were able to demonstrate 15 to 40 db recovery of hearing acuity over the frequency range tested during the first two weeks following the head blow. They also showed that deafness was more easily produced when the blows were delivered to the skull near the experimental ear. Gross and microscopic studies of the temporal bone and brain failed to reveal significant pathologic lesions in the middle ear and brain. With graphic reconstructions of the cochlea they found the primary injury to be a degenerative change in the organ of Corti with the upper part of the basal coil most susceptible to injury. They found, in the order of severity, these recognizable stages of end organ damage:

1. Loss of external hair cells.
2. Loss of external and internal hair cells.
3. Flattening of the organ of Corti.
4. Complete disappearance of the organ of Corti.

There was secondary degeneration of the nerve fibers and ganglion cells supplying the damaged regions of the cochlea. Perlman found that maximum nerve degeneration occurs three weeks after the head injury.¹⁰

According to Schuknecht²⁹ blows to the head create pressure waves in the bones of the skull which are transmitted through bone to the cochlea, where they produce a "shock pulse" in the fluids of the inner ear. This phenomenon is comparable to the effect of an airborne blast wave or intense noise on the tympanic membrane which is transmitted by the conductive mechanism to the inner ear fluids. These pressure waves displace the basilar membrane during analysis of the acoustic stimulation. Intense stimulation produces a violent displacement of the basilar membrane causing injury to the organ of Corti. Both reversible and irreversible cellular injury occurs in this end organ.

Schuknecht further observed that hemorrhage into the labyrinth did not appear to be responsible for deafness. "The location of blood had no correlation with the location of end-organ damage. In view of the findings it appears unlikely that blood cells act as an irritant in an intact labyrinth. Blood was found in the cochlea as long as four months after the head blow without causing reaction within the labyrinth."

INCIDENCE OF EAR INVOLVEMENT IN CRANIOCEREBRAL INJURY.

A series of 152 consecutive head injuries were examined by Grove⁶ within 24 hours. There was evidence of skull fracture in 17 cases and ear signs or symptoms in 49 cases (32.66 per cent). Mygind³ in otoscopic examination of similar cases found traumatic changes in about 50 per cent. In one-half, however, only a hyperemia of the drum was present. Mygind found evidence of vestibular trauma in one-third of the cases; Grove found an incidence of 26.6 per cent. Gurdjian³¹ reported a series of 718 acute cranial injuries seen during an 18-month period. Of these, 475 were proven to have skull fracture

(X-ray, inspection and autopsy). Bleeding from the ears occurred in 129 cases. Clinical evidence of brain damage was found in 246 cases. Alexander and Scholl³² reported demonstrable hearing losses in one-third of their reported series of 551 cases of head trauma.

In our present study of 100 consecutive cases of head trauma seen for otological evaluation, 98 revealed signs and symptoms referable to disturbances of structures contained within the temporal bone. It is our firm belief that the great majority of cranio-cerebral injuries will present subjective (tinnitus, vertigo, deafness, bleeding, headaches) or objective (unconsciousness, audiometric hearing loss, abnormal caloric test, spontaneous nystagmus, positional nystagmus, deformities of the external canal and drum, X-ray evidence of fracture, facial nerve paresis or paralysis) evidence of fracture or concussion.

OTOSCOPIC EXAMINATION.

A careful examination of the external canal and drum should be done as soon after the injury as possible. Lacerations in the skin may heal rapidly. Hematotympanum and liquor tympanum may be detected for only a few days. Fracture of the bony canal wall may be diagnosed by the presence of blood clots, or bloody crusts on its walls, or in the skin of the canal (ecchymoses), and by irregularities in the bony configuration of the canal. Deformities in the external canal wall were detected in eight of 28 longitudinal fractures in our series. The external canal is never fractured in pure transverse fractures.

BLEEDING FROM THE EAR.

Following a head injury bleeding may occur from the ear simply as a result of a laceration within the external auditory canal or from rupture of the tympanic membrane. In either event the amount of bleeding is minimal. If the hemorrhage is profuse it may be the result of injury to a large vein (lateral or superior petrosal sinus, jugular bulb), tympanic plexus of veins and arteries or the middle meningeal artery. Rarely is the carotid artery involved. If it is, there is profuse bleeding from the nose, mouth and ear. At times in spite of an

absence of a history of bleeding from the ear the examiner observes blood clots, ecchymosis or a hematoma on the walls of the external auditory canal, occurring most often on the posterior and superior walls of the canal.

Another sign of temporal bone fracture is the development of an ecchymosis over the mastoid area (Battle). This usually appears on the fourth or fifth day after injury.

Active bleeding in our experience usually stops spontaneously when the patient is placed in the upright position. It has never been necessary to expose the source of bleeding in patients with hemorrhage from the external auditory canal.

In transverse fractures bleeding into the middle ear is frequent without rupture of the tympanic membrane. This is discernible on otoscopic examination, and is called hematomypanum. The drum has a blue discoloration and bulges. Grove found this sign in seven cases. We observed this sign in two cases out of a hundred.

In Grove's 211 cases of skull fracture 112 bled from one ear and 34 from both ears. In 530 cases of skull fracture reported by Gurdjian³³ unilateral hemorrhage occurred in 281 and bilateral hemorrhage in 47 cases. In our study, which included 57 patients with skull fractures, bleeding occurred from the ear in 24 cases. Davis³⁴ reported a mortality of about 66 per cent in patients with bilateral hemorrhage, while in unilateral hemorrhage the mortality was 31 per cent. Bleeding from the ear is reported to occur in about 30 per cent of head injuries (24 per cent in our study).

Delayed or late hemorrhage from one ear may occur, and several such cases have been reported by Grove.³⁵ In two cases the hemorrhage occurred one month after the injury. This may have been due to involvement of a small vessel in an area of osteomyelitis or osteitis with erosion and delayed bleeding.

Fracture of the skull is frequently present in association with bleeding from the ear. Gurdjian reported 87 per cent demonstrable fractures in 129 cases of bleeding from the ear.³¹ Among the 84 cases with unilateral bleeding in this group there were 73 with positive Roentgen evidence of fracture and 11 with negative findings. In this same group the

site of the fracture was in the middle fossa in 48 cases, in the posterior fossa in 29 cases, and in the anterior fossa in 15 cases. In a recent study of Gurdjian and Webster³⁶ of 1285 cases of head injury there were 184 cases with bleeding from the ear and six cases with cerebrospinal otorrhea. Of the 184 cases with a bloody otorrhea 164 had a skull fracture—88 per cent. The mortality rate of those with bleeding from the ear was 17½ per cent, a figure much lower than in earlier reports, probably due to antibiotics and sulfonamides.

CEREBROSPINAL OTORRHEA.

The incidence of cerebrospinal fluid otorrhea has been reported by several authors. Besley³⁷ reported it in 20 patients among 1,000 with skull fractures; Yerger,³⁸ in 20 per cent of skull fractures; Grove,⁶ in eight of 211 cases. We found it in two of 57 cases with skull fracture. The series of 718 head trauma cases with 475 proven skull fractures reported by Gurdjian³¹ presented cerebrospinal fluid otorrhea in eight out of 129 cases with bleeding from one or both ears.

The otorrhea may be difficult to identify for several days because of the presence of blood; furthermore, the cerebrospinal fluid otorrhea may be scanty and of short duration so that it is not detected. On the other hand it may be copious (Ballance¹⁷ reported a case which yielded 600 cc. of fluid within 24 hours) and may persist for several weeks.

Grove⁶ felt that cerebrospinal fluid otorrhea is an indication of a longitudinal fracture of the temporal bone. Occasionally the onset of the cerebrospinal fluid otorrhea is delayed^{6,39,40}. This type of otorrhea has been observed behind an unruptured drum.^{41,42} This may be identified in an otoscopic examination by a clear fluid level in the tympanum.

Cerebrospinal fluid otorrhea usually subsides spontaneously and rarely requires surgical treatment. When it is accompanied by discharge of brain tissue, surgical treatment is indicated. The management is similar to that of an open skull fracture. We have had five instances of cerebrospinal fluid otorrhea and brain discharge from the ear during the past ten years. In such cases debridement with closure of the dural tear is

performed. If the dural tear is large a piece of temporal fascia or subcutaneous fascia from the scalp may be used for closure.

UNCONSCIOUSNESS.

In general, the more severe the injury the greater the likelihood of unconsciousness and the longer its duration. In Grove's series of 211 skull fractures involving the temporal bone there were 28 cases without unconsciousness. In our study of 100 cases of head trauma we found the following incidence of unconsciousness:

1. Transverse fractures, 16—unconsciousness, 9.
2. Longitudinal fractures, 28—unconsciousness, 9.
3. Skull fractures without temporal bone fracture, 13—unconsciousness, 3.
4. Head injury without fracture but with unconsciousness, 14.
5. Head injury without fracture or unconsciousness, 32.

TINNITUS.

Tinnitus is a common complaint after head trauma. It is not necessarily in proportion to the degree of trauma or of deafness and occasionally remains as the only residual disturbance.

In the 100 cases of *head trauma* in this study we found six of 16 cases of transverse fracture, seven of 28 cases of longitudinal fracture, three of 13 cases of skull fracture not involving the temporal bone, six of 14 cases of head injury without fracture but with unconsciousness, and 14 of 32 cases of head injury without fracture or unconsciousness, to have tinnitus.

DEAFNESS.

Head trauma frequently results in some degree of deafness. This may occur without the presence of a skull fracture, in which case it is commonly referred to as concussion deafness. A moderate blow to the head, which does not produce a fracture, will often produce a hearing loss varying from a mild dip at 4,000 cycles to severe inner ear deafness. The losses

are most severe for the tones between 3,000 and 8,000 cycles. In the severe cases the entire audible frequency range is usually involved with the greatest loss at the higher frequencies.

The hearing function is determined with the audiometer. Supplementary testing is done with the recorded voice tests and tuning forks. It is important to delay the testing until the sensorium is clear. Since both conductive and perceptive structures in the hearing mechanism are frequently affected by head trauma the audiogram taken shortly after the head injury may show a uniform hearing loss for all frequencies. Conductive lesions tend to resolve whereas perceptive lesions frequently do not or only partially recover so that the degree of hearing impairment will vary.

Audiometric studies on recovery have been reported by Schuknecht.¹³ Usually deafness is noted immediately after the injury, but it may not become manifest until later, when secondary degenerative changes in the cochlea take place producing a progressive deterioration of hearing. Loudness recruitment is present in perceptive deafness resulting from head injury. Since recruitment is absent in cochlear nerve lesions and present in Meniere's disease and noise deafness it strongly supports the concept that head trauma damages the organ of Corti.²¹

The tests for hearing are subjective, consequently one must be constantly aware of the possibility of malingering. Another important consideration is that there is usually no knowledge of the patient's hearing function before the time of injury. This is an important factor in medico-legal cases which involve settlements.

Saltzman¹⁴ believes that head trauma may damage the cochlear nuclei and secondary auditory tracts, resulting in a loss of low and high tones by air conduction. The bone conduction curve varies from frequency to frequency and does not follow the trend of the air conduction curve. He stated that this pattern appears several weeks after the head injury. We have attempted to evaluate the hearing loss in the following groups of head trauma cases:

1. Longitudinal fracture of the temporal bone.
2. Transverse fracture of the temporal bone.
3. Skull fracture without temporal bone fracture.
4. Head trauma with unconsciousness but without evidence of fracture.
5. Head trauma without unconsciousness or fracture.

Longitudinal Fracture of the Temporal Bone—The hearing was examined in 28 cases of longitudinal fracture. Three of these also had a transverse fracture, leaving a total of 25 cases with only longitudinal fractures. Of these nine had variable degrees of nerve deafness; 13 had mixed conduction and nerve deafness, and three cases demonstrated no hearing loss one to six weeks after the accident. Schuknecht⁴³ states that "the occurrence of conduction deafness alone in the fractured ear is uncommon. Some degree of inner ear deafness is almost always associated with the conduction loss." We found, too, as reported by Ruttin⁴⁵ and by Schuknecht⁴³ that when a hearing loss is demonstrated in the ear opposite the fracture it is of the inner ear type (concussion deafness).

The prognosis for recovery of hearing in longitudinal fractures is usually favorable. The conduction loss due to middle ear bleeding and trauma usually recovers. The perceptive loss in hearing in these cases has a poorer prognosis with a much less degree of recovery.^{2,6,8,43}

Grove⁶ stated that the function of hearing is more severely damaged in longitudinal fracture with increases in age; furthermore he tested the hearing in 108 of 112 cases of longitudinal fracture, and found loss of hearing in 79.5 per cent on the homolateral and 54.6 per cent on the contralateral side, indicating that labyrinthine concussion is of common occurrence in head trauma.

According to Skoog⁴⁶ longitudinal fractures present either a conduction deafness (65 per cent), perceptive deafness (22 per cent), or a mixed deafness (13 per cent).

Transverse Fracture of the Temporal Bone—In our study we had 16 cases all with total deafness on the affected side. On the contralateral side eight cases demonstrated a perceptive type of hearing loss. Grove⁶ reported 16 cases of

transverse fracture, all with homolateral complete deafness. He reported considerable damage to the contralateral ear in 65 per cent of these cases. Of interest are the cases reported by Kingenberg,¹⁶ and Nager⁹ in which fracture lines were visualized extending through the pyramid and in which vestibular function was intact but the cochlear function lost.

Skull Fracture Without Temporal Bone Fracture—A total of 13 such cases was examined. A bilateral nerve type hearing loss of variable degree was present in six cases. Two cases presented a unilateral nerve deafness, one of which was severe. Grove⁶ reported 49 such cases. In testing the 98 ears of these cases he found 45 with indication of inner ear damage. Even after eliminating malingerers and those with a hearing loss before injury, Grove felt that in many of these ears the loss in hearing was due to injury either from stretching of the cochlear nerve (side motion of the brain according to Ulrich²) or from hemorrhage into the inner ear. We believe that the hearing loss is due to the shock pulse injury to the organ of Corti as described by Schuknecht.⁴³

Head-Trauma With Unconsciousness but Without Evidence of Fracture—A total of 14 cases were examined in this group. Four had normal hearing; eight had a high tone loss and two presented a low tone loss. The group is small, but it suggests a rather high incidence of inner ear damage even though there is no clinical evidence of skull fracture.

Head Trauma Without Fracture or Unconsciousness—A total of 32 cases were examined. Normal audiograms were obtained in seven cases. A mild degree of bilateral nerve deafness was present in 12 cases. In five cases a severe degree of bilateral nerve deafness was present in two of which there was a previous history of acoustic trauma, and in one case central nervous system syphilis was uncovered. Four cases presented a unilateral total loss of hearing. Two cases had a previous conduction deafness due to chronic middle ear disease.

DISTURBANCES OF EQUILIBRIUM.

True vertigo and dizziness are common sequelae of head injury. These symptoms may arise from disturbances in the

vestibular sense organs, the primary vestibular nuclei and probably the central vestibular pathway.⁴⁷ In a subconcussion, vestibular disturbances may be absent, but in a severe concussion these disturbances are common.

Two types of vestibular disturbances may occur as a result of head trauma: one is the peripheral type, the result of derangement of the vestibular end-organ; the other, the central type due to derangement of the primary vestibular nuclei and central vestibular pathways.

The peripheral type usually presents a true turning vertigo (staggering, room movements) in short attacks. Spontaneous nystagmus, with the quick component to the opposite side, and disturbance in balance and past-pointing may be noted. Accompanying involvement of the autonomic system may be present with nausea and vomiting; perspiration with a cold, clammy skin; a rapid pulse and lowered blood pressure. These effects are probably mediated through connections of the vestibular nuclei with the vagus. Asymmetrical caloric response may be present. This type of vertigo usually subsides in several weeks. The transitory nature of the symptoms has been explained by the ability of the vestibular nuclei to compensate for the unbalanced impulses which one deranged labyrinth may initiate.

McNally⁴⁸ has indicated that dizziness which arises from a disturbance in the central nervous system may be difficult to differentiate from an attack of peripheral labyrinthine dizziness. If tinnitus and hearing loss are combined with true vertigo in short attacks and autonomic disturbances are present, the likelihood of a peripheral origin is more certain.

Errors of sensation (postural vertigo) such as tilting of the bed, a rising of the floor, a falling of the ceiling of a room, a feeling of uncertainty or of being drunk, were believed by Grove^{6,49} to be of possible vestibular origin if they were accompanied by an abnormal caloric response. Postural vertigo may be due to derangement in the utricular macula as a result of the pressure pulse transmitted to the vestibule as a result of a head blow. The utricular macula presents a gelatinous mass on its surface which contains calcium carbonate crystals. This mass (otolithic membrane) may be displaced by the

forces of linear acceleration and gravity. A head blow can conceivably injure this delicate mechanism.²¹

The central type of vestibular disturbance is usually a sensation of disequilibrium and not a sensation of true rotation. It is a common accompaniment of head injury. The term "blacking out" is sometimes used by patients to describe the attacks which may be initiated by sudden movements, such as stooping, straining, head turning and upward movement of the eyes. Rowbotham⁵⁰ attributes these momentary black-outs to instability of the cerebral circulation consequent upon injury to its vasomotor apparatus, rather than to labyrinthine dysfunction. Thus, he believes, a momentary ischemia of the brain, due to circulatory lability, is the essential cause of the dizziness. The usual disequilibrium seen in patients with head injury is more suggestive of brain stem involvement with implication of not only vestibular fibers but also of tectospinal, rubro-spinal and spino-cerebellar pathways.

The peripheral vestibular elements may be injured directly by the pressure pulse set up in the labyrinthian fluids by the head blow. Such cases are known to have hemorrhages into the inner ear fluids which may be a cause for disturbed vestibular function.²⁷ A more important factor may be injury to the nuclei and central pathways in the brain stem.

Recent work of the mechanism of concussion reveals that in closed head wounds, pressure gradients at the cranio-spinal junction result in shear and tear of the elements in the brain stem. Even subconcussive blows in dogs resulted in some cellular changes of the medulla, pons and midbrain. These reversible changes (subconcussive blows) may occur in the cells of the brain stem explaining the usual findings in the human case of concussion.⁵¹ Histologic changes have been demonstrated in the vestibular nuclei of animals after concussion injuries.⁵²

Concussion is defined as a head trauma with unconsciousness at impact due to involvement of the brain stem reticular formation, associated with a shock-like state and slow pulse. Subconcussion is a head trauma not severe enough to cause post-traumatic unconsciousness but nevertheless associated with some involvement of the reticular formation.

The vertigo which follows cerebral concussion without deafness appears to be explainable by the alterations in the central vestibular nuclei.^{47,52,53} That involvement of the brain stem pathways is important in the development of dizziness is suggested by the relative absence of dizziness in open wounds of the head where dissipation of injury at and about the open wound results in little or no involvement of the brain stem centers.

That disequilibrium may be indicative of a derangement in the central nervous system is suggested by Leidler⁵⁴ who lists the following symptoms as strongly suggestive of a central lesion:

1. Normal hearing combined with either attacks of vertigo or hyper-excitability of one labyrinth or spontaneous nystagmus;
2. Headache and vertigo;
3. Positional nystagmus;
4. Spontaneous nystagmus without vertigo;
5. Preponderance of the slow phase of the nystagmus; and
6. Rhythmic disturbances in vestibular reactions.

The following symptoms were listed by Van Egmond, Groen, Hulk and Jongkees⁵⁵ as proof of a central origin:

1. Spontaneous nystagmus towards a dead labyrinth or spontaneous nystagmus without diminution during a week;
2. Purely rotational nystagmus of long duration;
3. Vertical nystagmus;
4. Normal turning reactions without caloric excitability;
5. Monophasic reactions without quick phase; and
6. Somnolence in deviations of the positions of the head.

The dizziness consequent to head trauma is usually positional in character and varies in severity. Positional nystagmus is a spontaneous nystagmus which is not constantly present but develops when the head assumes a particular position in space.⁵⁶ The various positions used in the test have to be assumed rapidly in many instances of head trauma before the nystagmus is elicited. Like the caloric test, responses to positional testing are likely to vary on repeated examinations in these cases of head trauma. The positional tests may have to be repeated a number of times before the symptom is produced and the nystagmus seen.

In head trauma cases demonstrating a positional nystagmus and, especially when the hearing is not impaired, a positional nystagmus of variable direction is seen (Type I of Nylen and Lindsay).^{57,58,59,60}

Murphy⁶¹ found 32 cases of positional nystagmus in 176 cases of head trauma. He found that the results varied on repeated examinations. He felt that this test is valuable when positive, since it is never positive in normal persons and left no question of normal limits to be considered.

The direction-changing nystagmus frequently elicited in head trauma on postural testing suggests a disturbance in the vicinity of the vestibular nuclei.⁶⁰ According to Lindsay the vestibular centers may be considered as normally in a state of equilibrium. When a lesion occurs in the vicinity of the vestibular nuclei the ability to compensate for positional changes may be impaired.

Cupulometry has recently been reported to offer quantitative data on the function of the vestibule with respect to nystagmus and the sensation of vertigo.⁵⁶ This is a turning test with small regulable stimuli of a magnitude provoked by ordinary life movements. The subject is rotated at specified speeds of turning, and the duration of the resulting nystagmus is noted. At the same time the subjective duration of the sensation of vertigo is recorded. The duration of nystagmus and sensation of turning are plotted on a logarithmic scale. The resulting graph is the cupulogram. According to Jongkees, in many cases cupulometry has been the only examination which made the clinical picture clear, or gave an objective basis for subjective complaints.⁶²

The nystagmus and vertigo seen in the acute stage of a total loss of unilateral vestibular function (transverse fracture) are severe. The direction of the horizontal nystagmus is toward the side of the normal ear, the subjective vertigo in the direction of the nystagmus and the pastpointing opposite to the direction of the vertigo. These spontaneous vestibular symptoms subside within a month after destruction of the end-organ so that in the chronic stage spontaneous vestibular symptoms are absent.

Spontaneous nystagmus, vertigo and reaction movements may be severe in longitudinal fractures and even when a fracture cannot be demonstrated. The intensity of the vestibular symptoms is not a reliable guide for predicting the extent of the damage to the vestibular end-organ.⁶³ The degree of recovery varies a great deal and progressive deterioration may continue for many months. The vertigo varies in severity, and the vestibular responses likewise may be impaired to a varying degree.⁶¹ In patients with a dead labyrinth there is considerable disability even though the other labyrinth does in time compensate. Patients with such a disability are subject to errors in the sense of location in space when in the dark. Sudden changes in position may bring on an attack of vertigo.

We have attempted to analyze the equilibratory disturbances in the following group of head trauma cases:

1. Longitudinal fracture of the temporal bone.
2. Transverse fracture of the temporal bone.
3. Skull fracture without temporal bone fracture.
4. Head trauma with unconsciousness but without evidence of fracture.
5. Head trauma without unconsciousness or fracture.

Longitudinal Fracture of the Temporal Bone—The caloric test was performed in 17 of 28 cases of longitudinal fracture. A normal response was obtained in nine cases. A complete loss of vestibular function was observed in four cases: two of these had combined longitudinal and transverse fractures, a third had a permanent high tone deafness, and the fourth had a temporary high tone loss which eventually recovered. A hypoactive caloric response was observed on the side of the longitudinal fracture in four cases.

Spontaneous nystagmus, generally mild, was observed in seven of the 28 cases. This sign was observed as late as 10 months after the injury. Dizziness was complained of in 12 cases.

Positional nystagmus was observed in three cases; two with direction fixed, and one with varying direction.

Transverse Fracture of the Temporal Bone—Vestibular reaction to the cold caloric test (ice water) was absent in all 16 cases of transverse fracture. A weak spontaneous nystagmus to the uninvolved ear was elicited after head shaking in seven cases. Eleven of the sixteen cases complained of some disturbances in balance.

Skull Fracture not Involving the Temporal Bone—Out of 13 such cases a normal caloric test was observed in seven; two cases presented a hypoactive caloric response; five complained of dizziness; three had a spontaneous nystagmus; and two cases had positional nystagmus, one direction fixed and the other with varying direction.

Head Injury With Unconsciousness—Fourteen cases were seen in this group. A normal caloric test was obtained in nine cases. An asymmetrical (hypoactive) response was obtained in four cases. Dizziness was present in six of the cases; one was severe. A faint spontaneous nystagmus was observed in three cases. Objective, positional nystagmus was observed in three cases; two were direction fixed and one varying direction. Three additional cases presented a history of bouts of dizziness with change in position, but the positional nystagmus could not be elicited.

Head Injury Without Unconsciousness or Fracture—A total of 32 cases were seen. The caloric test was normal in 16 cases and asymmetrical in five cases. Dizziness was a symptom in 16 cases, four of which were severe. Dizziness was absent in 13 cases. A spontaneous nystagmus was observed in three cases. Positional nystagmus of fixed direction type was observed in two cases.

SUMMARY OF VESTIBULAR FINDINGS.

One hundred cases of head injury were reviewed. Fifty-seven of these had a skull fracture; 50 complained of dizziness; 76 cases had a caloric test. The caloric response was abnormal in 35 cases and normal in 41 cases. Positional nystagmus was observed in 10 cases out of approximately 40 tested.

The abnormal caloric tests consisted for the most part of hypoactive or absent responses on the involved side. Hyperactive responses were not observed. Positional nystagmus

was "fixed" in seven of the 10 cases, where it was observed indicating a peripheral vestibular disturbance. In three cases it was of the variable direction type, suggesting a central disturbance. Neurological examination in these three cases failed to show other evidence of a central lesion.

Most of our cases were seen on only one occasion for examination, some many months after the head injury. Consequently, we were not in a position to have a complete clinical picture in each case. Our review showed only 10 per cent of the cases to have objective positional nystagmus. We believe the incidence is much higher.

Usually postural vertigo disappears after a week, or a few weeks. The same holds true for other findings in our review. The material in this study consists of 100 patients who complained of certain or all the symptoms of the vestibulo-cochlear mechanism. The discrepancies seen in the figures are due to the fact that all did not complain of all possible symptoms. Thus, the 32 patients without unconsciousness or fracture complained of dizziness in 50 per cent; of deafness in 75 per cent; and tinnitus in 43 per cent. It should be pointed out that these patients represent a convalescing group; thus they do not give a total cross-section picture.

ROENTGEN FINDINGS.

The visualization of basal fractures involving the petrous bone by Roentgen studies is often difficult. The presence of a radiating fracture line extending toward the top of the petrous bone may be considered as evidence of a longitudinal fracture of the petrosa. This is particularly true when there is bleeding from the ear. Special views such as Stenver's, Mayer's, Schuller's and Chamberlin's may be of value in demonstrating a temporal bone fracture. The recently developed "magnification" techniques⁶⁶ are helpful. In general, few longitudinal fractures involving the temporal bone can be shown in conventional Roentgen studies. The fresher the fracture the more likely will be its detection.

Transverse fractures are more readily visualized. Stenver's view is the projection of choice. This fracture can often be detected many years later because of the failure of the otic

capsule to heal, once it is fractured. In this series of 44 temporal bone fractures, 26 had positive Roentgen findings of a fracture. Eight of 16 transverse fractures and 18 of 28 longitudinal fractures were positive; the latter group were vault fractures extending into the base. Roentgen findings were positive in 89 per cent of Gurdjian's and Webster's 1285 cases of skull fractures.²⁶

TREATMENT.

Treatment in this class of patient includes the management of the bleeding ear, pneumocephalus, possible meningitis and mastoiditis.

The bleeding ear is best treated by the application of fluffy gauze to the external ear and a bandage to hold the gauze in place. Usually bleeding or cerebrospinal fluid otorrhea stops spontaneously in the course of one to five days. Patients with aural bleeding should be on prophylactic doses of sulfonamides and antibiotics to prevent infection, particularly meningitis.

Meningitis is a serious complication of temporal bone fracture, and was common before the days of sulfonamides and penicillin therapy. The fact that, in many, fractures remain patent for many years following injury, and the fact that these fractures may communicate with preformed pathways giving access to the intracranial spaces, threaten the development of intracranial infections many weeks or months after injury. This has definitely changed since the use of antibacterial therapy. Such patients who develop a meningitic attack have been successfully treated and cured, and this is the rule rather than the exception in this class of patient.

Mastoiditis and otitis media were also common complications following head injury before the days of antibiotic and sulfa therapy. These, too, have been greatly diminished. The presence of such complications should be noted and appropriate treatment given. Persistent mastoid suppuration in spite of antibacterial therapy would call for eradication of the infected bone. Suppuration in the air cells of the petrosa may require surgical exenteration of the involved cells. Prophylactic middle ear exposure is discussed by some otologists but this concept is not fully accepted.⁶⁵

THE FACIAL NERVE.

Incidence of Facial Nerve Injury—In closed head injuries Grove reported 29 cases of facial paralysis among 211 patients with skull fractures involving the ear. In our study of 57 fractures nine had facial paralysis. Gurdjian reported 14 cases of facial paralyses in 129 cases with bleeding from one or both ears, an incidence of about 11 per cent. Two of the patients in this group had bilateral facial paralysis, one survived and one died. The survivor has had complete recovery of function.

The paralysis was complete and permanent in one transverse fracture of the petrous bone and in five with fractures of a longitudinal type, according to the report of Grove. In the remaining 23, the paralyses improved or cleared up completely. Grove stated that all authors agree that facial paralysis occurs more frequently in transverse than in longitudinal fractures. The incidence in the former is approximately 50 per cent. In longitudinal petrous fractures the incidence varies from 10 to 20 per cent. In our series of 57 fractures, facial paralysis occurred once in 16 transverse fractures (recovered) and seven times in 28 longitudinal fractures (two permanent). In our own experience almost all of the patients with facial paralysis improve or clear up completely. The patients with permanent facial paralysis following craniocerebral injuries are rare. We have had only two such examples, one of which was treated by spinal facial anastomosis.

Facial nerve paralysis in lacerations of the face are seen in some cases of craniocerebral injury. We have operated on seven such cases. Four were in lacerations involving branches of the pes anserinus, and three were of lacerated facial trunks proximal to the pes or near the stylomastoid foramen.

Signs and Symptoms—A complete facial nerve paralysis involves an inability to contract the facial muscles of expression on the affected side; the patient is unable to whistle, or to wrinkle the forehead on the affected side. When he is asked to close both lids, the eyeball deviates up and out. The corneal reflex is lost on the involved side, which becomes of practical importance in testing lid reflexes in the comatose patient. If the paralyzed side only is examined, the incorrect conclusion may be reached that the coma is profound. Atrophy occurs as a late result, with a reaction of degeneration present

in the muscles of the face and with degeneration fibrillations recordable by electromyography. The progress toward recovery can be estimated by these methods. Rarely a bilateral complete facial paralysis occurs.

Facial paralysis of the peripheral type may occur immediately at the time of impact or it may be seen four or five days after injury. The type that begins several days after injury usually is recovered from quite satisfactorily.

It is important to identify a peripheral facial paralysis from a central type of facial paralysis. In the central type there is usually activity of the forehead and the eye, with serious involvement of the musculature about the mouth. Electrical reactions are also normal, but this is of little aid since the reaction of degeneration does not occur until about 14 days after the onset of the paralysis. This type of facial paralysis is, of course, on the contralateral side from the cortex or pyramidal system involved.

The facial nucleus in the pons may be damaged in head injury resulting in a unilateral peripheral type of paralysis. Other cranial nerve nuclei or central tracts may also be involved in such deep seated lesions, characterized by multiple intrapontile hemorrhages. Single massive hemorrhages also have been observed in patients who may have associated lesions, such as a subdural hematoma with uncal herniation resulting in anoxia with hemorrhages from increased permeability of the vessels.

Occasionally a masking of the face may be seen in the chronic stage of head injury due to a mimetic or emotional facial weakness. This may result from deep-seated damage, probably involving extrapyramidal connections with the facial nucleus, as may occur in Parkinsonism. The patients manifest diffuse brain injury associated with pyramidal tract signs, and may have survived a long unconscious state.

Pathology—The paralysis as stated previously may be immediate or may appear as late as several days to two weeks after injury. An immediate onset usually indicates physiological or anatomical nerve injury in the Fallopian canal, or at the internal auditory meatus. A delayed onset of the facial paralysis usually is due to an edema of the nerve associated

with the reparative processes at the fracture site. Small hemorrhages within the nerve sheath with edema may also be a cause for this condition. In many instances nerve function returns, and this has been attributed to recovery of the nerve from injury by stretching, contusion, interfascicular hemorrhage and even section. It appears possible that a torn facial nerve under favorable conditions may regenerate. Ischemia of the nerve from edema has also been suggested as a cause for the paralysis. Cases of bilateral facial paralysis are rare but have been seen, and might be more frequent, if some of the more seriously injured individuals survived long enough for careful neurological examination.

Recovery is almost always the end-result in patients with facial paralyzes due to closed head injuries of the head. At times there may be partial recovery with marked stiffening of the face on the affected side, and mass movements of the entire face such as in laughing or in expressive activities. This may be associated with evidences of twitchings and fibrillations of muscles in the facial group. If recovery is complete, it usually follows a short duration of facial paralysis. Facial paralysis which persists longer than three months usually will improve in part only. If the aforementioned, fibrillations and mass movements of the face on emotional activity are present and if recovery does not occur within 18 months to two years, one should consider the advisability of spinal facial anastomosis or hypoglossal facial anastomosis. We are not impressed that in the greatest majority of cases decompression of the nerve in the facial canal would be of value, since a majority of these patients recover completely, or almost completely, in a reasonable period of time, and results of improvement after operation should not necessarily be construed as evidence that the operation is effective.

Open Injuries—In craniocerebral injuries facial nerve paralysis due to laceration of the nerve on the face or near the mastoid area poses a definite problem.

The nerve or its branches may be involved anywhere on the face. Shell fragment wounds near the ear and the mastoid area may destroy a segment of the nerve, presenting little hope for reconstructive surgical treatment of the nerve. Damage following mastoid surgery may produce anatomical

destruction of the nerve trunk. Knife wounds, lacerations on the side of the face and injury associated with the removal of parotid tumors may cause extensive injury. Lacerations near the mastoid area may produce a simple division of the trunk, and in some instances there may be an involvement of the branches of the pes anserinus as well as the parotid duct.

Treatment—In closed head injury, treatment of facial paralysis of the peripheral type is always conservative, employing physiotherapy with massage, mild heat and exercise of the facial muscles. The prospect for recovery is promising in the greatest majority of cases. By means of the electrical responses of the facial muscles to faradic and galvanic currents, observations may be made which will aid in prognosticating the outcome or the need for further therapy. A period of about nine months should be allowed for spontaneous recovery. Operative intervention in our hands is not employed until 18 months have elapsed without any improvement in the facial function.

Open injuries, the result of shell fragments or bullet wounds, may be of such massive degree as to preclude repair of the facial nerve in any part of its course. Lathrop has found it possible to deal with this type of injury in some cases with considerable success by the use of nerve grafts.

Stab wounds and lacerations of the trunk or its branches offer the possibility of complete recovery of function when the nerve can be anastomosed. A most important requirement in this type of injury is exploration and suture of the nerve immediately after injury. This is true particularly of the branches of the facial nerve peripheral to the pes anserinus. When the branches have been sectioned, scar tissue usually makes identification impossible in late cases. When the parotid duct is found sectioned, the duct may be sutured about an inlying ureteral catheter. The catheter may be removed in a week. When a parotid fistula is present, complicating a wound of the facial nerve trunk, the use of Roentgen therapy to the parotid gland may be useful in preparing the wound for exploration. Secretions stop after several treatments.

When irreparable injury has involved the central portions of the nerve, the use of a facial hypoglossal or facial spinal accessory anastomosis is useful. A degree of tone develops in the facial muscles which encourages symmetry in the resting face. Voluntary movements of the face are dependent upon associated shoulder or tongue movements. Some patients become very agile in the use of the associated movements to bring about expressive changes of the face.

Nerve grafts may be considered in the treatment of cases not suitable for direct nerve anastomosis. Plastic procedures employing fascial slings have been found helpful, or a buccal fascial splint which is attached to the upper molar region on the paralyzed side may be useful.

SUMMARY AND CONCLUSIONS.

1. The types of blows commonly associated with fracture of the temporal bone are reviewed and classified. Temporo-parietal area blows spread the skull in an antero-posterior direction producing a vertical fracture in the lateral aspect of the skull which may extend toward the base of the skull, producing a longitudinal fracture of the temporal bone.

2. Occipital area blows spread the skull from side to side fracturing it vertically near the midline. The fracture may extend forward to the middle fossa and fracture the petrosa transversely, producing a loss of cochlear and vestibular function and frequently damage to the facial nerve.

3. Frontal area blows also spread the skull from side to side fracturing it vertically near the midline. The fracture may extend posteriorly to the base of the skull in the anterior, middle, and even posterior fossae, resulting in a transverse fracture of the petrosa.

4. The types of blows producing isolated fractures of the base are reviewed.

5. The pathologic findings in longitudinal and transverse fractures are summarized.

6. Early experimental and pathological studies of temporal bone fractures emphasized hemorrhage into the labyrinth as the primary cause of cochlear and vestibular disturbances. Recent investigations suggest that neural degeneration in the

membranous labyrinth is due to shock or a pressure pulse set up in the inner ear fluids by the head blow. Intense stimulation produces a violent displacement of the basilar membrane, causing injury to the organ of Corti. The same stimulus may disrupt the otolithic membrane on the end-organs of the utricle and saccule resulting in disturbances referable to those sense organs. Hemorrhage into the labyrinth in the light of recent studies does not appear to be responsible *per se* for deafness or for vertigo.

Hearing losses following head trauma are rarely if ever attributable to central lesions. Bilateral destruction of the auditory cortex does not create a threshold loss for hearing. Up to 75 per cent of nerve fibers to a particular region of the cochlea can be destroyed without creating threshold hearing losses for frequencies localized in that region, and the constant presence of loudness recruitment supports the concept that head trauma damages the organ of Corti.

7. Repeated audiograms are necessary to properly assess the end result of head trauma on hearing.

8. Dizziness following head trauma may be either peripheral or central in origin. In transverse fractures there may be a severe vertigo resulting from destruction of the membranous labyrinth. Postural vertigo is believed to result from injury to the utricular macula and its associated otolithic membrane. The central type of dizziness is a sensation of disequilibrium, and not one of true rotation. The term "blacking out" is the most descriptive. It is suggestive of brain stem involvement (vestibular nuclei and fibers, tectospinal, rubro-spinal and spino-cerebellar pathways).

9. In closed head wounds pressure gradients build up at the craniospinal junction resulting in shear and tear of the elements in the brain stem. In open head wounds the forces of injury are quickly dissipated, the brain stem is usually not injured, and dizziness does not usually occur.

10. The symptoms and findings in central involvement of the vestibular mechanism are reviewed.

11. Postural vertigo and positional nystagmus are frequent findings in cases of head trauma. Injury to the peripheral vestibular mechanism results in a positional nystagmus of

fixed direction. A direction-changing nystagmus suggests a disturbance in the central vestibular nuclei and pathways.

12. Cupulometry has recently been reported to offer quantitative data on the function of the vestibule with respect to nystagmus and the sensation of vertigo.

13. The findings on clinical examination of head trauma cases are often temporary. Many times these cases are seen for only one examination. It is only with repeated and thorough examination that a complete clinical picture is obtained.

14. A thorough and painstaking Roentgen examination is required to demonstrate temporal bone fractures. The recently developed magnification techniques are recommended where available.

15. Treatment of temporal bone fractures is conservative. A prophylactic use of antibiotics and sulfonamides is advisable. Aural bleeding can often be controlled by merely having the patient assume a sitting position. Threatening suppurative complications may call for appropriate surgical intervention.

16. Most cases of facial paralysis clear up spontaneously. Surgical intervention is seldom necessary. Treatment is conservative with physiotherapy, massage and exercises.

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SIALOGRAPHY IN THE DIAGNOSIS AND TREATMENT OF LESIONS OF THE PAROTID GLAND AND DUCT.

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Since the publication of a paper on sialography¹ by one of us ten years ago, the employment of this procedure has frequently facilitated the diagnosis and treatment of lesions of the salivary apparatus. The technique as there described has required no essential modification; however, valuable experience has been gained in the interpretation of Roentgen shadows, especially those characteristic of uric acid calculi.

Parotid injection is a comparatively simple procedure and apparently devoid of danger. We have on occasion seen the duct perforated and lipiodol injected into the surrounding tissues without harmful results.

Injection is best performed by reflected light with the patient reclining upon the X-ray table. A 5 cc. glass syringe fitted with a blunt 3/8 inch 20 gauge needle simplifies the procedure. In order to decrease the viscosity of lipiodol, the "loaded" syringe with needle attached is allowed to remain in a hot water bath until the time of injection. Steno's duct, which makes an almost right angle turn as it passes over the masseter muscle, is now straightened by pulling the cheek outward and forward. The mouth of the duct is then dilated by means of No. 1 and No. 2 lachrymal probes, followed at once by the slow injection of 1. - 1.5 cc. of warm lipiodol. A small wad of cotton is placed over the papilla so as to prevent leakage and confusing shadows. Roentgenograms are taken in lateral, oblique, and P. A. positions.

Interpretation of sialograms may require some training, but with a little practice characteristic shadows may be ob-

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served in uric acid calculi, fistulae, strictures, sialectasis, mixed tumors, malignancies and extra-glandular lesions; but, the most practical value of sialography lies in differentiating mixed and malignant tumors, in the accurate determination that malignant degeneration of a mixed tumor has taken place.



Fig. 1. Normal sialogram. Note uniform caliber of duct.

Note in the sialogram of a normal parotid, the uniform caliber of the duct (see Fig. 1.) and the arborescent character of the gland parenchyma (see Fig. 2). In the oblique position an accessory gland is clearly visible (see Fig. 3). In mixed tumors the duct is apt to be displaced forward and downward (see Fig. 4) due to what the radiologist calls a space-occupying mass. The substance of the gland, however, fills perfectly.

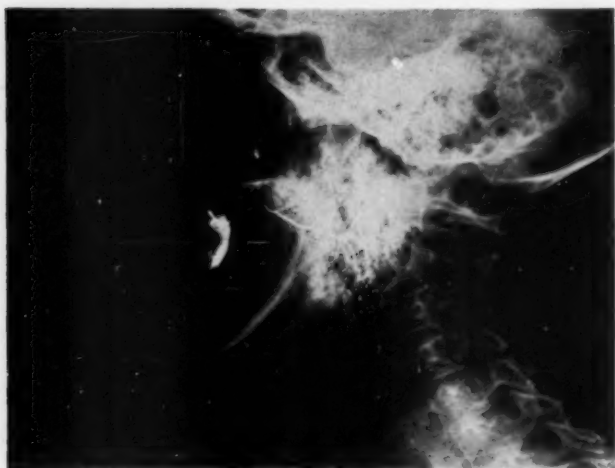


Fig. 2. Same as Fig. 1. Note arborization of gland parenchyma.

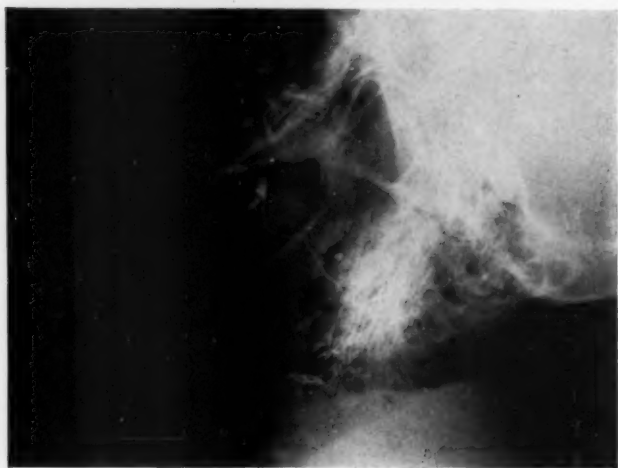


Fig. 3. Same as Fig. 1. Accessory parotid gland above Steno's duct.

In malignancies the glandular parenchyma is destroyed completely (see Fig. 5). In such cases the accessory gland may greatly hypertrophy and lead to error in diagnosis.

Besides facilitating the differential diagnosis of salivary lesions the injection of iodized oils has at times proven to be of definite therapeutic value. Idiopathic swellings of the parotid not infrequently subside promptly. Whether this is



Fig. 4. Mixed tumor. Note: (a) Hairpin curve of Steno's duct, due to "space-occupying mass" pushing duct forward. (b) Accessory parotid and duct just above Steno's duct. (c) Normal filling of gland parenchyma.

due to bactericidal action, the increased flow of saliva or lubrication has not been determined, but the fact remains that following this simple procedure parotid swellings often vanish readily and permanently.



Fig. 5. Sialogram of Fig. 12. Malignant mixed tumor. Note parenchyma of main gland destroyed. Accessory gland greatly hypertrophied.



Fig. 6. Salivary fistula due to razor cut. Repair followed by retention catheter. Improved method of anchoring catheter described in text.

The treatment of lesions in and about the parotid has been materially advanced by sialography and the use of retention catheters. Aside from infectious parotitis the most common lesions encountered are calculi, mixed tumors, fistulae, strictures and malignancies in the order given. The surgical treatment of calculi² and malignancies of the duct³ have been described elsewhere.

Masseteric parotid duct fistulae (see Fig. 6) may be readily closed by isolating and carefully coapting the cut edges of the

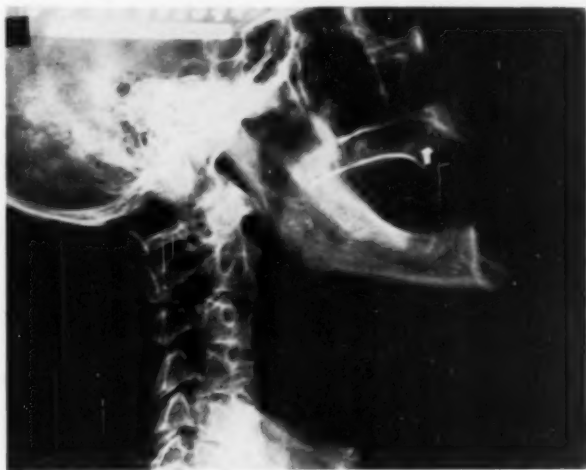


Fig. 7. Catheter in Steno's duct fixed to cheek by means of skin clip.

divided duct with fine catgut and inserting a retention catheter (see Fig. 7) which is allowed to remain *in situ* for two to three weeks. Re-establishing the normal channel in this manner has distinct advantages over older, more uncertain, methods of converting external into internal fistulae.

Strictures of the duct (see Fig. 8) which do not respond to dilatation may be dealt with in a similar manner. The constricted portion of the duct is resected, and the freshened proximal and distal ends are united by fine catgut sutures, followed by retention catheter.

Following the extraction of ductal stones, too, the retention catheter has proven to be an excellent prophylactic measure against the development of strictures.

Care must be exercised not to use too large or too long a catheter which might cause pain and irritate the tail of the gland. A No. 5 ureteral catheter, $2\frac{1}{2}$ inches long seems the

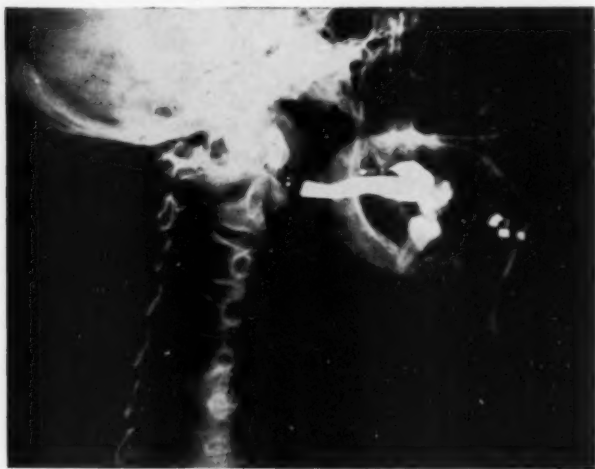


Fig. 8. Dilated Steno's duct of long duration due to stricture. (Probably stone.) Stricture resected, followed by catheter.

most practical. By passing the end of the catheter through the eye of a medium sized skin clip and anchoring this to the buccal mucosa it causes no discomfort and may be left *in situ* indefinitely.

Mixed tumors, due to their anomalous character, pose a singular problem. Mixed tumors are made up of cartilaginous, endothelial and epithelial elements. As many as 25 per cent are said to develop malignant characteristics, rupture their capsule and invade surrounding structures. The causes of this change are not apparent but evidence is accumulating that radiation, trauma, and repeated surgical efforts are

responsible. The common tendency of radiating all salivary tumors should, therefore, be strongly deprecated.

Mixed tumors grow slowly. They are always encapsulated and may attain great size (see Figs. 9 and 10). Because of



Fig. 9. Mixed tumor of the parotid. Pueblo Indian, showing size mixed tumor may attain without malignant degeneration.

the danger of malignant change all mixed tumors should be carefully extirpated. In small or moderately sized tumors, an incision made over the most prominent part, in the line of the facial nerve fibers or Steno's duct, will rarely injure



Fig. 10. Calcifying muco-epidermoid adenoma of right parotid.



Fig. 11. Malignant mixed tumor. Squamous-cell carcinoma Grade 4. Radical resection, 1941. Patient survived 8 years in spite of refusal of neck dissection.

these structures; nor is there much danger of salivary fistula in carrying the incision right through the substance of the gland. To safeguard against this eventuality a catheter may be left *in situ*. The accessory parotid must, of course, be identified and avoided.

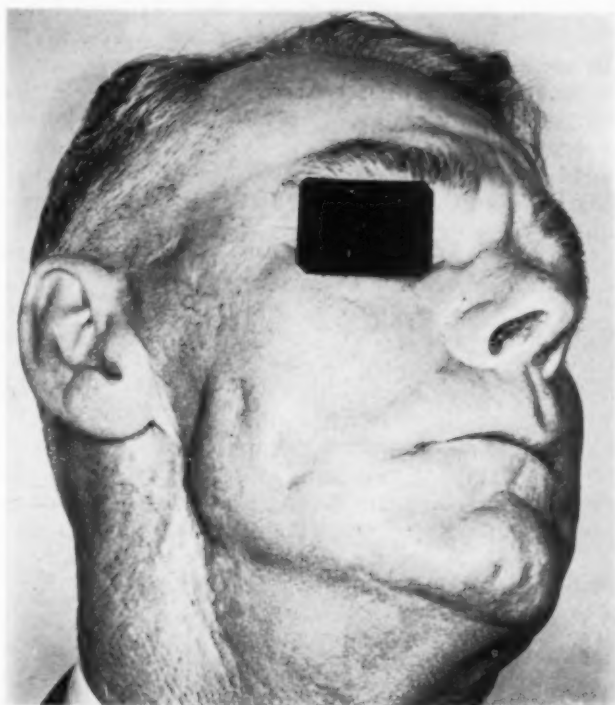


Fig. 12. Parotid duct calculus, mixed tumor and malignancy in the same patient. Radical resection in 1949. Scars in cheek due to fascial implants (elsewhere) which became infected. Patient well since December, 1949.

Before leaving the subject of mixed tumors, attention should be drawn to an article published in the *Archives of Otolaryngology* (Aug. 1953). Mumma⁴ reports the case of a mixed tumor of the tongue successfully treated by the injection of colloidal chromic phosphate ($\text{CrP}_{32}\text{O}_4$). "There has been no

recurrence in three years." This is an encouraging report that augurs well for the treatment of these lesions by radio-active chromic phosphate.

Malignant tumors of the parotid (see Figs. 11 and 12), whether primary or secondary, generally demand radical resection with sacrifice of the facial nerve. Prolonged efforts calculated to preserve the integrity of this structure appear to us as non-surgical. Radical parotid surgery may be a life-saving measure. The resulting facial paralysis is clearly outweighed by the patient's chances of recovery; besides, fascial implants are now successfully employed with excellent cosmetic results. Without going into the details of technique it may be noted that ligation of the external carotid greatly facilitates the operation.

In the employment of contrast visualization techniques preliminary to radical surgery of the parotid a word of caution may not be amiss. The value of sialography as an operative determinant must not be overrated. Deliberate sacrifice of the facial nerve is probably never justified by Roentgen findings alone; fortunately, in most instances, other signs and symptoms are present which corroborate the diagnosis. Sudden rapid growth and pain in a dormant mixed tumor are highly significant. Infiltration, local fixation of the skin and cervical adenopathy are ominous signs which suggest the true nature of the lesions. Facial paralysis attests that the nerve has become infiltrated by the malignant process.

CONCLUSIONS.

Sialography has become a valuable adjunct in the diagnosis of lesions of the salivary apparatus.

In idiopathic swellings of the parotid the injection of iodized oil may be of great therapeutic value.

The use of the retention catheter is of value in the repair of salivary fistulae and strictures as well as in the prevention of ductal strictures.

The greatest value of sialography lies in differential diagnosis of mixed and malignant tumors.

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- 227 16th Street.
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SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY
AND OTOLARYNGOLOGY.

Arrangements have been completed for the joint meeting of the North Carolina Society of Eye, Ear, Nose, and Throat, and the South Carolina Society of Ophthalmology and Otolaryngology September 17, 18, 19, 1956. Headquarters will be the George Vanderbilt Hotel, Asheville, North Carolina.

An unusually attractive program has been arranged, and a large attendance is anticipated.

Asheville, North Carolina, is in the mountains of Western North Carolina, and is a particularly beautiful spot in this season of the year.

For further information write Roderick Macdonald, M. D., Sec. and Treas., 330 East Main Street, Rock Hill, S. C.

STRICTURE OF THE EXTERNAL AUDITORY CANAL.

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The purposes of this paper are to present the information gained from the study of eight cases of acquired closure of the external auditory canal and to describe a simple endaural technique for the correction of this deformity. Numerous papers^{1,2,3,4,5,6} concerned with agenesis, or aplasia of the ear canal, have appeared in the otolaryngologic literature, but few recent publications have dealt primarily with closure of acquired origin.

On occasions, the nomenclature of the two entities has been confusing. House⁷ agreed with Kinney⁸, who referred to congenital absence of the meatus as "aplasia", and felt that the term "atresia" should be reserved for acquired stenosis. "Atresia", however, is a Latin word; but it is derived from two Greek words, "A" and "tretos". The latter means "to perforate". The literal translation, then, of "atresia" is "against perforation" or "without perforation," and would not necessarily indicate a canal which had been once open and subsequently closed. Perhaps the word "stricture" from the Latin "stringere"—to draw tight, is more apropos for the abnormality under consideration.

Cohen and Fox⁹ believed that ear canal stricture was rare, having observed it in only two out of 17,000 cases, in which care of the ear, nose and throat was sought. Ballenger and Ballenger¹⁰ suggested ear canal cartilage atrophy, with subsequent wall collapse, as an occasional cause. Novick¹¹ reported a case which was probably due to the careless use of caustics or curettes. Two cases of stenosis, accompanied by mastoiditis and cholesteatoma, were reported by Lillie and McBean;¹² and they advocated surgery to prevent intracranial complications in such cases. The use of split thickness skin grafts with sponge packing, to ensure patency, was suggested by Conley¹³.

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* From the Department of Otorhinolaryngology of the University of Kansas School of Medicine.

ANALYSIS OF CASES.

The Chief Complaint.

Eight cases of true meatal stricture were encountered in our outpatient department in the past three years. Two patients complained of postauricular fistulae, one of headache on the involved side, one of vertigo, three of pain in the ear, and one of cosmetic deformity. None have hearing loss as the chief complaint, although all admitted to it on questioning. The duration of difficulty varied from six months to 19 years. One had been previously operated without success.

The Cause.

Three of the strictures were produced by burns—one chemical and two thermal. One was the result of a football injury, and one followed a razor wound. Two were due to surgical procedures performed for the removal of parotid cysts or tumors, and one was incident to an automobile accident.

Radiologic Findings.

In seven cases mastoid destruction to some degree was demonstrable by X-ray, while the uninvolved side invariably exhibited normal pneumatization. An area of rarefaction in the region of the antrum, suggesting cholesteatoma formation, was a surprisingly common feature (five cases).

Hearing Loss:

Since most of the patients were suffering from infection and bone destruction, it was felt that the hearing was of secondary importance; nevertheless, air and bone conduction determinations were done on all cases. As one might expect, those of several years' duration possessed hearing curves typical of mixed type deafness. Those of more recent origin showed a conductive type loss, but the hearing in every involved ear was below the 30 db level for all tones in the conversational range. Two older individuals had high tone loss on the uninvolved side.

Physical Findings.

With one exception there was complete fibrous closure of the canal in the outermost portion. In one instance it was

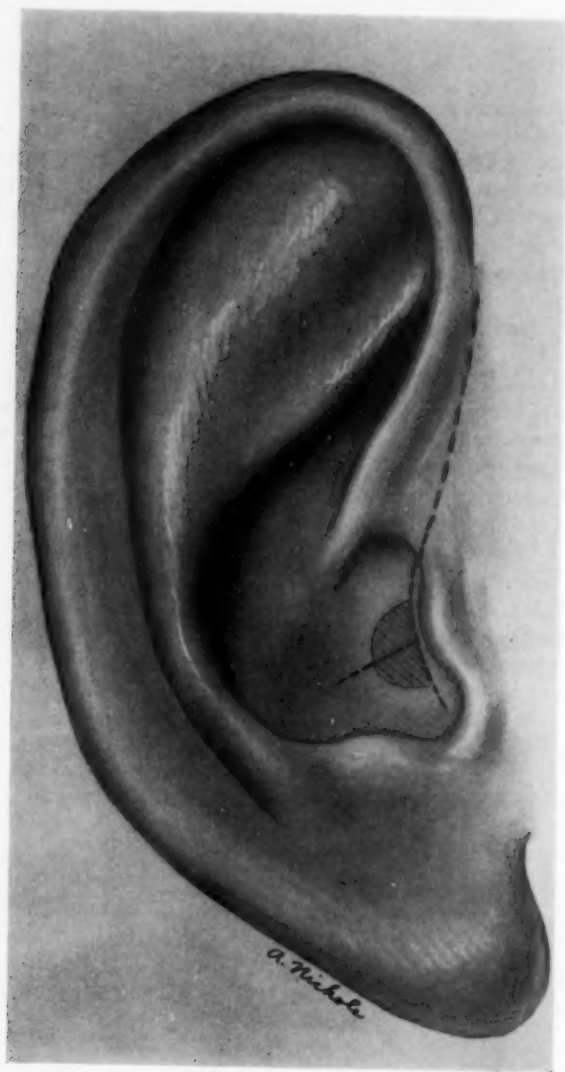


FIG. 1. The Incision.

possible to pass a small probe into the lumen for at least one cm. A probe could also be introduced into the postauricular fistulae previously mentioned, and cortical dehiscences were thus palpated.

The patient, who had sustained the football injury, had also suffered loss of the lobule. Retroauricular tenderness was present in two of the cases, and in one of these, nystagmus of the second degree was demonstrable. The neck of one individual was badly scarred as a result of the same burn which was the genesis of the stricture. Otherwise, all were in reasonably good health.

Treatment.

All eight cases were treated surgically by a technique which embodied certain of the principles used by both Cohen and Fox, and Conley, yet it differed in some respects from the operative pattern followed by any of these authors. The steps of the operation were as follows:

1. A skin incision was made just anterior to the helix and parallel to this structure. It extended from the superior point of attachment of the helix to a position just posterior to the superior margin of the tragus.

2. Hugging the posterior margin of the tragus, the same incision was extended inferiorly to the region where the inferior wall of the ear canal would ordinarily be.

3. From the center of the post-tragal portion of the above incision another incision was made in a posterior direction and at right angle to the first. It terminated at a point where the posterior canal wall would ordinarily be (see Fig. 1).

4. Two triangular flaps of skin and underlying fibrous tissue were thus created, one above and one below; and the subcutaneous tissue was resected from these flaps (see Fig. 2).

5. Any existing mastoid or middle ear disease was then eradicated by the customary endaural method.

6. A No. 000 silk suture was introduced postauricularly opposite the superior triangle of skin, brought out through the base of the triangle, reintroduced into its apex and re-

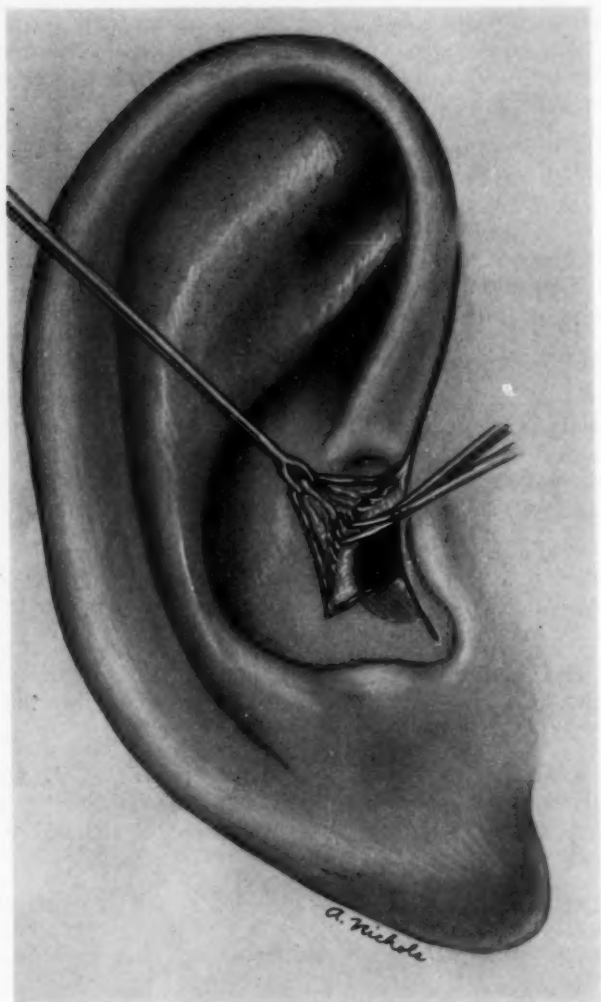


Fig. 2. Removal of Subcutaneous Tissue.

turned to the postauricular area about 0.5 cm. superior to the point where it first entered the skin. The two ends were then

tied without tension, thus approximating the superior flap in juxtaposition to the canal. This suture emulated the one used by Sooy¹⁴ to maintain patency of the meatus in the fenestration operation.

7. A second postauricular suture, similar to the first, was used to apply the inferior flap to the posteroinferior canal wall (see Fig. 3).

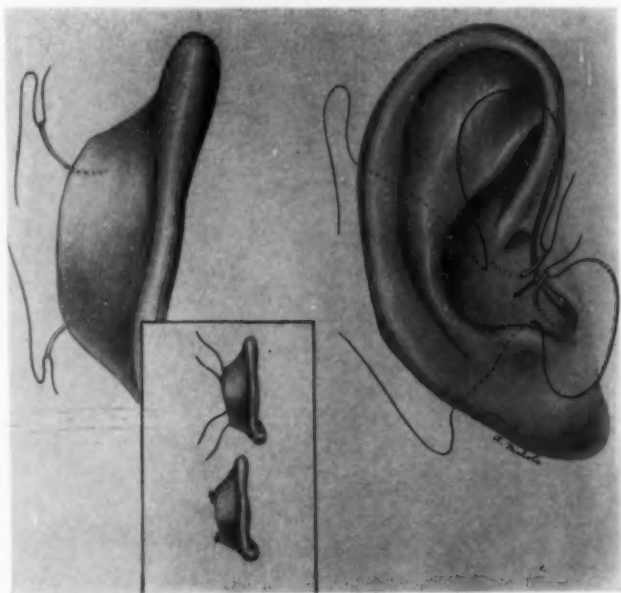


Fig. 3. Suturing.

8. A split thickness skin graft, obtained from the upper thigh, was applied to the mastoid and middle ear cavities and packed firmly in place with continuous grease gauze.

9. The supratragal portion of the incision was closed with interrupted No. 00000 silk sutures (see Fig. 4).

10. Packs and sutures were removed on the sixth postoperative day.

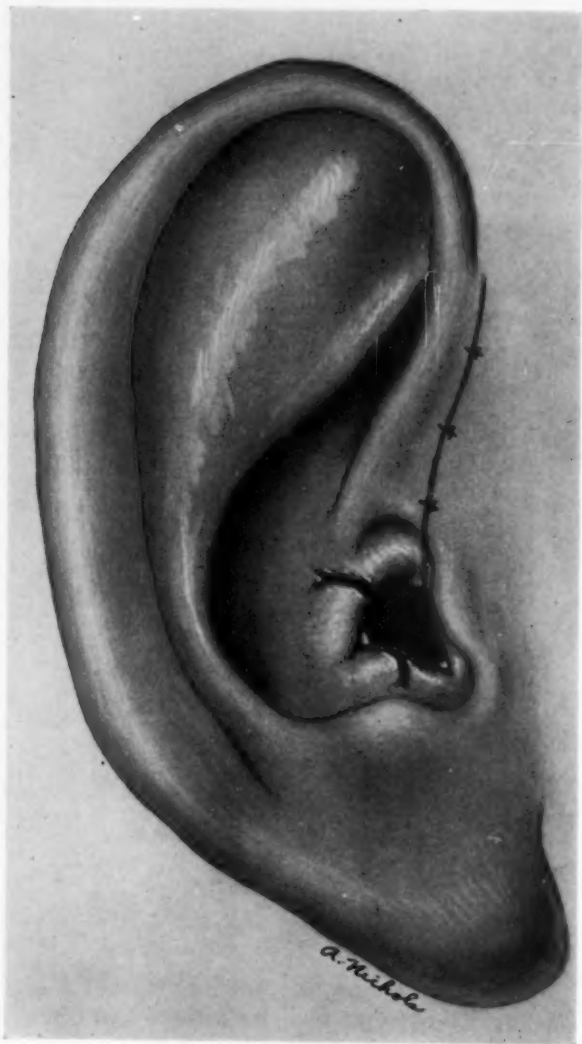


FIG. 4. Incisions Closed.

Operative Findings.

In most cases the extent of mastoid and middle ear disease was surprising, for no case was free of infection. Cholesteatoma was present in three canals, and there were cortical dehiscences in two instances. A fistula was present in one horizontal semicircular canal. Wide dural exposure was evident in one operation, and it amounted to almost complete absence of the tegmen mastoidea.

There was only one example of minimal infection, and that was apparent in the case of only six months' duration. Here, the membrana tympani was still intact, and the mastoid process was not diseased; nevertheless, the canal was full of pus, and it was assumed that more serious trouble lay ahead.

Results.

Permanent patency of the external auditory canal was obtained in all patients, and the cosmetic results were invariably good. Six of the ears became completely dry, but two still form granulation tissue and mucoid discharge. Both of the cases with postauricular fistulae were troublesome.

Although each fistula was carefully closed at the time of surgery, both reopened within two weeks. Secondary closure by marginal skin excision, undermining and suturing was successful. Improvement in hearing was realized by only one patient, for in all others the condition had been present for so long that extensive middle ear disease had developed.

ILLUSTRATIVE CASE.

J. B., a 63-year-old colored male, was first seen in the outpatient department on Dec. 30, 1953. He stated that he had sustained a severe burn of the left side of the face in 1938, and from that time on the ear canal had progressively narrowed. It finally closed entirely in 1948, but on occasions a bead of foul smelling pus would appear. Severe aural pain began two months prior to his outpatient visit.

The past history included mild attacks of asthma of 20 years' duration, and was otherwise irrelevant.

The only significant finding on physical examination was complete fibrous stricture of the left external auditory meatus, associated with moderate scarring of the auricle (see Fig. 5). X-ray revealed destruction in the mastoid process and an area of rarefaction anterior to the lateral sinus (see Fig. 6).

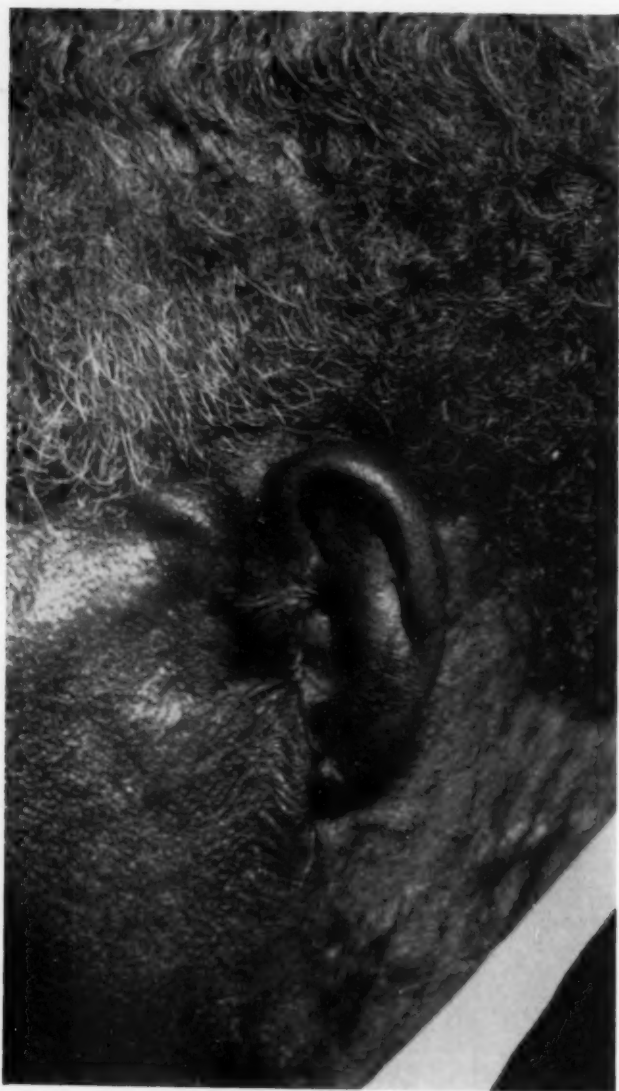


Fig. 5. Preoperative Stricture.

An X-ray of the chest revealed a fibrotic lesion of the right apex. This was diagnosed as quiescent tuberculosis, since on repeated sputum examinations no tubercle bacilli were found. The EKG presented no abnormality, and all laboratory findings were within normal limits.

On March 2, 1954, he was admitted to the hospital, and surgery was performed two days later. The procedure was identical to that described in the context, and pus and necrotic bone were found in the mastoid process. A modified radical mastoidectomy was done, and the mucocutaneous flap was used to cover the medial antral wall. A split thickness skin graft, prepared from the upper thigh, was applied to the temporalis muscle, tegmen mastoidea and facial ridge. The skin incision was closed after packing the cavity with aureomycin ointment gauze.



Fig. 6. Mastoid X-ray.



Fig. 7. Postoperative Result.

The postoperative course was uneventful, and the pack and sutures were removed on the seventh postoperative day. All discharge had ceased six weeks later and canal patency has been maintained until the present time (see Fig. 7).

DISCUSSION.

Appraisal of the operative findings would seem to indicate that any acquired external auditory canal closure may well be dangerous to the patient. The acquired type is probably more apt to be so, because the object which causes the wound will usually introduce infection into the canal or middle ear. Unless associated with a primary cholesteatoma, aplasia of the ear canal is less hazardous unless a previous unsuccessful attempt at surgical correction has been undertaken.

It would appear that radiographic studies of the temporal bone are of great value, not necessarily to establish the presence or absence of an ear canal, but also to determine the existence and extent of mastoid disease.

The endaural repair offers the advantages of simplicity and good cosmetic result, and it provides an epithelial lining of the meatus; furthermore the lines of stress, so created, favor permanent patency.

CONCLUSIONS.

1. Mastoiditis, middle and inner ear infection and postauricular fistula are complications of stricture of the external auditory canal.
2. The deformity deserves early surgical correction.
3. An endaural approach, resulting in a complete epithelial canal lining, may be utilized.
4. New lines of stress will aid in maintaining canal patency.

SUMMARY.

An analysis of eight cases of stricture of the external auditory canal is presented, and an endaural approach for the correction of the condition is described.

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**DALLAS ACADEMY OF OPHTHALMOLOGY
AND OTOLARYNGOLOGY
PROGRAM 1956**

Tuesday, February 7, 1956

Amon Carter Airport—Private Dining Room

Joint Meeting Fort Worth E.E.N.T. and Dallas O. and O.

Dr. Frank B. Walsh

Baltimore, Maryland

4:00 P.M.—Program:

Tumors of the Optic Nerve and Chiasm

5:30 P.M.—Cocktails, courtesy Alcon Laboratory

6:30 P.M.—Dinner honoring Dr. Walsh

7:30 P.M.—Myasthenia Gravis

THE DIAGNOSIS AND TREATMENT OF UPPER
RESPIRATORY SYMPTOMS PRODUCED BY
INCREASED VISCOSITY OF MUCUS AND DRYNESS OF
THE MUCOUS MEMBRANES.*

JACK R. ANDERSON, M.D.,

and

WALLACE RUBIN, M.D.,

New Orleans, La.

Because otolaryngologic thinking is so oriented in terms of allergy and infection, there is a tendency to overlook the fact that in approximately 20 per cent of the cases, symptoms accompanying upper respiratory disease are caused by increased viscosity of mucus and varying degrees of dryness of the mucous membrane instead of hypersecretion or abnormal fluidity as is generally assumed.

The true nature of the condition can be established by taking a good history, performing a thorough physical examination, and conducting a cytologic study of the secretions.

The purpose of this presentation is to review the diagnosis of this condition and to report the use of intranasal histamine in its treatment.

SYMPTOMATOLOGY.

Table I is a compilation of the more frequently encountered complaints due to increased viscosity of the mucus and varying degrees of dryness of the mucous membrane of the upper respiratory tract. We have used verbatim quotation to emphasize how truly common these symptoms are in the average otolaryngological practice, and to call attention to the fact that they may strongly mimic those due to diametrically opposed conditions, *viz.*, hypersecretion, or abnormal fluidity.

* From the Ear, Nose, and Throat Allergy Clinic of the Eye, Ear, Nose, and Throat Hospital, New Orleans, Louisiana.

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TABLE I.

FREQUENTLY ENCOUNTERED SYMPTOMS DUE TO INCREASED
VISCOSITY OF THE MUCUS, AND VARYING DEGREES OF DRYNESS
OF THE MUCOUS MEMBRANE OF THE UPPER
RESPIRATORY TRACT.

A. NASAL SYMPTOMS:

1. Blockage of the nose.
2. Dryness inside the nose.
3. Burning sensation in the nose.
4. Tickling in the nose.
5. Sinus trouble.
6. My nose is always blocked and nose drops give me relief for only a short time.
7. The only relief I get for my nose blockage is to wash it out every day with salt water.
8. The only way I can get relief of my nasal blockage is to have my nose packed.
9. I don't sneeze often, but when I do and my nose runs a little, I feel much better.
10. A big glob of mucus accumulates in my nose, and I feel terrible until I can work it loose.
11. At times my nose gets dry and starts to bleed a little.

B. OROPHARYNGEAL AND PHARYNGEAL SYMPTOMS.

1. I have a dripping from the back of my nose into my throat.
2. I have to "hawk" a great deal to clear the back of my nose.
3. I think my palate has fallen; or, I feel as though that little thing hanging from my palate is too long and is lying on my tongue.
4. I have to spit up a lot of thick phlegm every morning.
5. I have a lot of thick mucus in my throat, which causes me to gag, and sometimes even vomit when I try to remove it.
6. My throat gets awfully dry.
7. My mouth gets awfully dry; or, my mouth is so dry that I feel as though it's full of cotton.
8. Every morning when I first awaken I have a sore throat which passes off after I am up and about for a while.
9. I have a burning pain in the tongue; or, my tongue gets awfully sore and sensitive at times.
10. I have a metallic taste in my mouth.
11. I have a lump in my throat; or, the muscles of my throat seem to get tired easily; or, I have pain in my throat when I swallow; or, there is a painful spot inside my throat.

C. LARYNGEAL SYMPTOMS:

1. I have to clear my throat constantly.
2. I feel as though there is something down in my throat, but I can't seem to get it up; if I could, I think that I would feel better.
3. I get hoarse off and on, even though I don't have a cold.

D. CHEST SYMPTOMS:

1. Cough.
 2. I often get a burning sensation in the middle of my chest when I take a deep breath.
 3. I hear a wheezing in my chest, particularly when I lie down at night.
 4. I seem to get short-winded very fast; I must have something wrong with my lungs or my heart.
-

TABLE II.

SOME CAUSES OF INCREASED VISCOSITY OF THE MUCUS AND VARYING DEGREES OF DRYNESS OF THE MUCOUS MEMBRANE OF THE UPPER RESPIRATORY TRACT.

<i>Etiologic Agent:</i>	<i>Mechanism Postulated:</i>
1. Vitamin-A deficiency.	1. Epithelial change.
2. Nicotinic acid deficiency.	2. Epithelial change.
3. High carbohydrate intake.	3. Increased mucin content.
4. Fear and anxiety.	4. Vasoconstriction due to increased sympathetic tone.
5. Smoking.	5. Same due to nicotine.
6. Excessive coffee drinking.	6. Same due to caffeine; dehydration caused by diuretic effect of caffeine.
7. Chilling of the body surface.	7. Reflex vasoconstriction.
8. Ingestion of iced-beverages.	8. Reflex vasoconstriction.
9. Post-menopausal (natural or surgical).	9. Loss of trophic effect of sex hormones on mucosa.
10. Diabetes mellitus.	10. Same as No. 3.
11. Hypothyroidism.	11. Increased sensitivity to endogenous epinephrine.
12. Hyperthyroidism.	12. Increased tissue metabolism.
13. Excessive perspiration.	13. Dehydration.
14. Diarrhea.	14. Dehydration.
15. Vomiting.	15. Dehydration.
16. Fever.	16. Dehydration.
17. Post-alcohol ingestion.	17. Dehydration.
18. Diabetes insipidus.	18. Dehydration.
19. Nephrotic syndrome.	19. Dehydration.
20. Insufficient intake of water.	20. Insufficient hydration.
21. Acute and chronic infection.	21. Change in membrane.
22. Belladonna or other parasympathetic blocking agents.	22. Removes parasympathetic antagonism and leaves sympathetics unopposed.
23. Diuretics.	23. Dehydration.
24. Ephedrine, amphetamine, benzedrine, and other sympathomimetic drugs.	24. Sympathetic preponderance.
25. Antihistaminic drugs.	25. Parasympathomimetic action; abolishes antagonistic action of histamine to epinephrine.
26. Oxygen inhalation.	26. Dehydration.
27. Anatomic abnormalities of the external nose, septum, turbinates, nasopharynx, new growths, foreign bodies.	27. Cause abnormal negative pressure, air streams, interfere with ciliary activity, stagnation of secretions.
28. Surgical removal of important structures.	28. Removal of mucus producing structures.
29. Mild allergic states with any of the factors named herein superimposed.	29. Decreases water content of mucus.
30. Lowered atmospheric humidity.	30. Decreases water content of mucus.
31. Central heat without proper humidifying apparatus.	31. Decreases water content of mucus.
32. Air-conditioning.	32. Reflex vasoconstriction.
33. Inhalation of drying or astringent dusts.	33. Drying of mucus.

ETIOLOGY.

Some of the causes of increased viscosity of the mucus and varying degrees of dryness of the mucous membrane of the upper respiratory tract are listed in Table II.

PHYSICAL FINDINGS.

The color of the mucous membranes varies from an angry, dark red to a very pale pink, the so-called normal color range being seen at times.

There may be an appearance of normal moisture, or the mucosa may be so dry and the light reflex so dull that one almost expects to see cracks in it. Sometimes small crusts of dried mucus will be found on such membranes.

In some instances, no evidence of mucus is present other than the impression gained from the glistening appearance of the membrane; however, when a cotton-tipped applicator is lightly passed over the mucosa, a thin layer of very viscid mucus will be observed. The surface tension of this material is so great that it is often very difficult to remove it for transfer to a microscope slide for examination.

At times, mucus threads joining adjacent structures will be encountered. Small clumps of globules are frequently found. If these occur on the vocal cords, they may be confused with small polyps or nodules, but, of course, they will disappear after the patient has coughed several times. Large accumulations of mucus will be found in the presence of anatomic obstruction.

The mucus is usually clear in color but becomes more opaque as the viscosity increases. When it has been present on the membrane for a prolonged period, it may become somewhat yellow in tint. If the condition is due to excessive smoking, it may be stained by the nicotine and tars from the tobacco. At times, it may be blood-streaked, particularly after nasopharyngeal or laryngeal "hawking".

Just as any shade of membrane color may accompany this condition, so any degree of membrane engorgement may be present. In some instances, the turbinates will shrink normally when a vasoconstrictor is applied, while in others there

will be little change. This is some indication of the submucosal tissue changes which have occurred.

CYTOLOGIC FINDINGS.

Cytologic study of the mucus is an indispensable aid in the diagnosis of this condition.

The first characteristic noted is in the preparation of the material for staining. Because of its increased viscosity, one has difficulty spreading it into a thin layer with the usual match stick or wire applicator. Then, a long time is required for drying prior to fixation with heat. To speed the process, we sometimes direct a stream of compressed air onto the smear.

During staining with Hansel's stain, decolorization is rather difficult, and frequently one finds it necessary to apply alcohol several times before a sufficient amount of methylene blue has been removed. Even after extensive decolorization royal blue will be the predominant color when the stained smear is held up to be examined by daylight.

Upon examination of the stained smear, several findings are noteworthy. There is usually a relatively large amount of homogeneous-looking mucus containing few cellular elements; however, small accumulations of leucocytes and/or eosinophiles may be found in varying proportions in certain areas, and it is not unusual to see a surprising number of non-lobulated leucocytes; this latter phenomenon we have considered an indication of chronicity. The thicker the mucus, the more epithelial cells will be found in the smear. Quite often one will encounter a great many bacteria. This may indicate either a subacute infectious process or stagnation of secretions.

TREATMENT.

In a recent consideration of one aspect of this problem¹, a rather dismal outlook was given as far as therapy was concerned. We are not in agreement with this hopeless attitude, and feel that otolaryngologists have much to offer these patients.

Definitive treatment is most to be desired, but until this

can be accomplished, or if it cannot be achieved, the patient should be given the benefit of symptomatic relief. We shall consider only symptomatic therapy in this presentation.

Symptomatic treatment may be directed towards any one or more of the following:

1. Moistening the mucous membranes.
2. Liquifying the viscid mucus.
3. Stimulating mucus production by systemic administration of drugs.
4. Stimulating mucus production by local application of drugs.

The efficacy of treatment will depend largely upon the amount of metaplastic change which has occurred in the mucosa.

1. *Methods of moistening the mucous membranes.* These range from the use of a steam kettle to moisten the inspired air to douching the nose with a saline solution several times daily. Patients also use oils and petrolatum to achieve this effect. Some individuals use the common vasoconstrictor preparations for this purpose, although they erroneously believe them to be effective because they shrink the mucous membranes. These methods are not satisfactory, because of the short-lived relief of the symptoms that they provide.

2. *Methods used to liquify viscid mucus.* Anything that moistens the membranes will, to some extent, also liquify viscid mucus; however, certain mucolytic agents are available. These include: five per cent aqueous solution of ammonium chloride, Alevaire (R), a saturated solution of potassium iodide given orally, and intravenous sodium iodide.

3. *Mucus-stimulating materials administered systemically.* In addition to the aforementioned iodides, any adrenergic blocking agent may be used. We have used Hydergine (R) to great advantage in this connection^{2,3}. The dosage is one sublingual tablet four to six times daily. At times, it is necessary to reinforce the action of Hydergine (R) by the concomitant administration of 15 grains of prostigmine bromide. One may take advantage of the side actions of Rauwolfia

serpentina or chlorpromazine to stimulate production of turbinate engorgement and mucus production. All of these agents will give a certain degree of central sedation.

4. *Mucus-stimulating materials applied locally.* Proetz⁴ recommends a solution of five per cent ethyl alcohol, three per cent glycerine, and 0.9 per cent sodium chloride in distilled water to stimulate the nasal glands and to retain moisture. The use of 0.25 cc of a 1:2000 solution of prostigmine methyl-sulfate sprayed into each nostril four times a day has been advocated by Soskin and Bernheimer.⁵ Glas⁶ employed histamine iontophoresis in increasing doses, beginning with a solution of 1:2000 and reported excellent results. Van Dellen, Bruger and Wright⁷ used strong solutions (25 per cent) of acetyl-beta-methyl-choline with some success. We have used nasal sprays and drops of histamine in dilutions varying from 1:2500 to 1:5000 in those cases without too-far advanced metaplasia. The solution can also be used in the form of Dowling packs in the office. Their application results in engorgement of the turbinates and an increased flow of mucus of thin consistency.

If the patient is allergic, an attack of sneezing will be precipitated when the solution is introduced into the nose. It is rather dramatic to use such preparations in wide-open nasal passages which the patient believes are blocked. Within a few minutes there is a diminution in the volume of the lumen, yet the patient states that he feels a great deal better.

DISCUSSION.

It has been said⁷ that "careful investigations by able workers have left the cause of most nasal troubles still in doubt." This is not entirely correct. What is true is the fact that much of the basic work has been ignored, and our thinking has been too heavily oriented in terms of hypersecretion as a manifestation of infection, allergy, or the need for surgical intervention. In short, we have been too prone to think in terms of overfunction of the respiratory mucosa as a cause of symptom production. Scant attention has been given to "underfunction" as a basis of symptoms. Undoubtedly, we have been abetted in this error by the fact that patients have a tendency to be etymologically sloppy in their selection of

words to describe symptoms. As an example, they may often use the word "blockage" when "stiffness" would be more accurate.

SUMMARY.

1. A number of symptoms commonly encountered in the practice of otolaryngology are considered in terms of increased viscosity of mucus, and varying degrees of dryness of the mucous membranes of the upper respiratory tract.

2. Treatment based on this concept is discussed.

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705 Baronne Building.

CONGENITAL PERICHONDritis ASSOCIATED WITH ACHONDROPLASTIC.*

ROBERT C. KRATZ, M.D.,

Newport, Ky.

At the Cincinnati General Hospital I recently had the opportunity to examine and treat two cases of perichondritis auricularis, which, in my opinion, were congenital. Since I can find nothing in the literature on congenital perichondritis auricularis, these two cases are reported here.

An achondroplastic dwarf was born March 3, 1954, at the Cincinnati General Hospital. This child was the sixth offspring of normal parents. The other five siblings were all healthy and normal. On May 13, 1955, 14 months later, a second achondroplastic dwarf was born to the same parents. These two siblings had identical skeletal deformities, but the second child had a severe cleft palate, while the first had only a bifid uvula. Both of these infants developed bilateral serous perichondritis auricularis. The older of the two developed the condition in the fourth week of life, while the younger infant developed the effusion during the second week. These two children were treated differently and with varying degrees of success.

Case 1. The older child was treated by aspiration of a clear yellow fluid and the application of a collodion cotton mold, which was removed after six days. Three days later, however, the child was returned because the effusion recurred. Again a cotton collodion mold was applied, and not removed for 15 days. Following the second removal there was no recurrence, and the result was excellent. The duration of the disease by this treatment was 41 days.

The younger child was treated by repeated aspirations of the fluid and applications of a wet cotton mold. These aspirations were repeated every three days, and a wet cotton mold

* From the Department of Otolaryngology at the University of Cincinnati.
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Fig. 1. Case 2. The second and youngest of the two achondroplastic infants. This photo was made at one week of age. The ears were normal when this photo was taken.

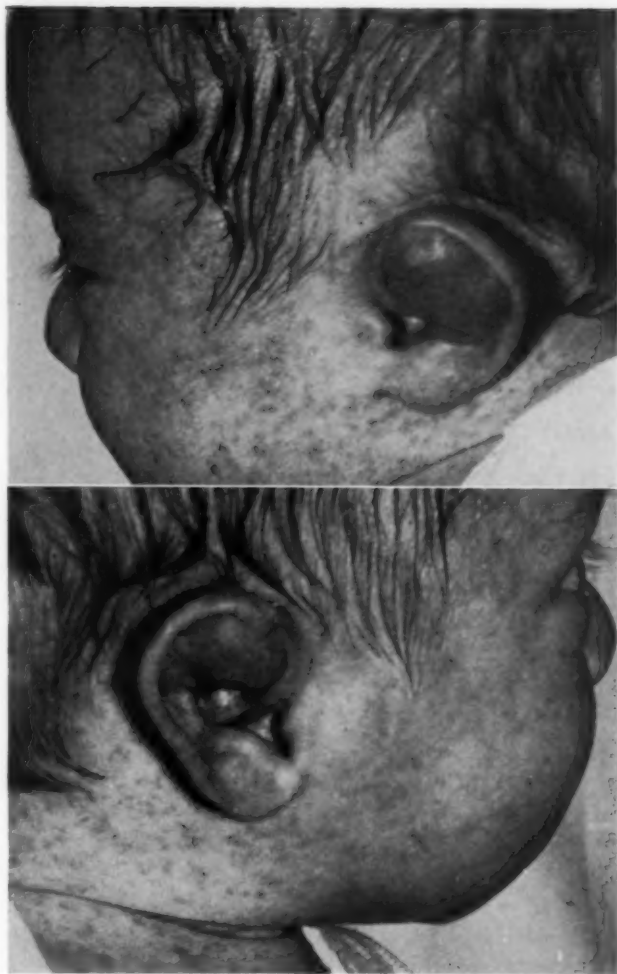


FIG. 2. These photos illustrate the right and left ears of the second of the two siblings at the age of five weeks. The preceding siblings had the same appearance at approximately the same age.



Fig. 3. The second of the two siblings was treated with wet cotton molds. The above photos illustrate the poor result from this treatment. The above photos were taken at the age of two months. The older sibling, who was treated with an ear mold, had an almost perfect result in contrast to the pictures.

reapplied. After two-and-one-half weeks of treatment the condition had not improved. The swelling extended to the place of closing the external auditory meatus. A cotton colodion mold was then applied. This was changed three times before the condition subsided. The result was guardedly fair. The ears were thickened and slightly deformed. The duration of the disease was 68 days.

Discussion:

Since 1941 there have been four achondroplastic dwarfs born at the Cincinnati General Hospital, including the two just mentioned. It could not be determined whether the other two children developed perichondritis auricularis, but since there is no mention made in their charts of the condition I assume they did not.

Judging from my limited experience with two cases of this rare condition, it would seem that the treatment of choice is to aspirate the fluid and fit an ear mold, which is held in place by an elastic type bandage, and is not removed until it becomes loose. It would seem also that treatment should be carried out for a month or longer.

SUMMARY.

Two cases of presumed congenital perichondritis auricularis occurring in achondroplastic siblings are presented.

Suggestions for the treatment of congenital perichondritis auricularis have been offered.

Third and Washington Streets.

In Memoriam

JOHN MACKENZIE BROWN, M.D.

1878 - 1955

John Mackenzie Brown was born in London, Ontario, Canada, in 1878. He received his preliminary education and was graduated from medical school at the University of Western Ontario in 1899. Shortly thereafter, he decided to come to the Southwest and made his permanent home in Los Angeles at the turn of the century, becoming an American citizen within a few years. Becoming interested in otolaryngology early in his career, he decided to obtain special training in this field and traveled extensively studying at the University of Michigan, University of Pennsylvania, Harvard, Edinburgh, and at several European centers.

His skill in his special field was quickly recognized, and he was appointed to a number of hospital staffs in Los Angeles. He was one of the first members of the staff at Children's Hospital, and remained active in that institution from 1909 until 1955. Not only did he serve as Chief of the Department of Otolaryngology, but also served as Chief of Staff at that hospital for a number of years. He maintained affiliations with many other hospitals in the city, including St. Vincents Hospital, the Eye and Ear Hospital, Hospital of the Good Samaritan, and the Cedars of Lebanon Hospital. His services as a consultant were constantly sought at these and other hospitals.

With the re-opening of the School of Medicine at the University of Southern California in 1928, Doctor Brown became vitally interested in the welfare of this institution and became Professor and Head of the Department of Otolaryngology in 1930. Several years later he became Chief of the Department of Otolaryngology at the Los Angeles County General Hospital, and even after his promotion to Professor Emeritus in 1950, he maintained an active interest in the work of both institutions.

The Residency Training Program and the Postgraduate Education in Otolaryngology were greatly stimulated and put



JOHN MACKENZIE BROWN, M.D.

1878 - 1955

on a solid footing during his period of leadership. His Wednesday rounds at 1:00 o'clock became traditional at the County Hospital and were renowned throughout the country for their excellence and stimulation.

Doctor Brown was deeply interested in the national otolaryngological societies, and served as an officer in many of them. In addition to serving as President of the American Laryngological, Rhinological and Otological Society, and President of the American Academy of Ophthalmology and Otolaryngology, he was also a member of the Los Angeles Society of Ophthalmology and Otolaryngology, the Pacific Coast Oto-ophthalmological Society, the American Otological Society, and the American Laryngological Association. He was a devoted member of all of these organizations and rarely failed to attend a national or local meeting of any society in the field of otolaryngology.

During his many years in the practice of otolaryngology, he contributed much to the advancement of the specialty and his contributions in several fields are world famous. His work on the frontal sinus, including his advocacy of early trephine in acute frontal sinusitis and conservative surgery in chronic frontal sinusitis; his work in branchial-cleft anomalies and his interest in intracranial complications were well recognized throughout the world. He served as Associate Editor of the Archives of Otolaryngology for many years.

All of these sterling qualities and great accomplishments in the field of medicine do not tell the real story of "J.M.", a truly beloved physician. He will be remembered by many thousands of patients who knew him as a kind and able physician with real devotion to the sick. To his fellow physicians, he was known as an astute diagnostician and a very able surgeon in the field of otolaryngology. To many otolaryngologists now practicing, not only on the West Coast but also in various other parts of the country, Doctor Brown will always be the great teacher, the great educator, and the kindly advisor who guided and helped innumerable young men in the specialty of otolaryngology. He was deeply interested in people and in education. He was a strong guiding force in

the early days of organization of the School of Medicine at the University of Southern California, and for many years was a member of the executive committee of the faculty. A large group of his former students and friends have for years met at the annual meetings of the American Academy of Ophthalmology and Otolaryngology as the "John Mackenzie Brown Alumni Group", and have awarded an annual recognition to that member of the group who made the best contribution to the literature in that year. This award is considered a great honor. In 1949, a testimonial dinner was given to Doctor Brown by a large number of otolaryngologists in the Southern California area upon the occasion of his completion of fifty years of practice. At this testimonial dinner, an oil painting was presented to him by his devoted friends, and copies of this painting hang in many otolaryngological offices on the West Coast.

"J.M." was a most humble and modest person who always tried to remain quietly in the background; nevertheless, his great character and human qualities were so evident that many honors were presented to him during his years in practice.

Remaining actively engaged in practice and in his educational work until the very end, Doctor Brown passed away in his sleep on December 31, 1955. He is survived by his widow, Marian, two daughters, two sons, and six grandchildren.

The John Mackenzie Brown Memorial Fund has been established to perpetuate the name of the man who was known as the dean of otolaryngologists on the West Coast, and who made contributions to the specialty, which will be permanent in the archives of otolaryngology. This fund will be used to build a lecture hall in the new University of Southern California Medical School Center. This will be a fitting memorial to Doctor Brown, who was very anxious to see education in medicine raised to the highest possible level.

Contributions to the University of Southern California Medical School, attention John Mackenzie Brown Memorial Fund, may be forwarded to the fund office, Suite 308, 1136 West Sixth Street, Los Angeles 17, California. H. H.

NATIONAL SOCIETY MEETINGS.

Schedule of Meetings for 1956:

American Board of Otolaryngology, to be held at the Sheraton-Mt. Royal, Montreal, Canada, May 6-11.

American Otological Society, Inc., to be held at the Seignior Club, Ottawa, Canada, May 11-12.

American Laryngological Association to be held at the Seignior Club, Ottawa, Canada, May 13-14.

American Broncho-Esophagological Association, to be held at the Sheraton-Mt. Royal, Montreal, Canada, May 15-16, (afternoons).

The American Laryngological, Rhinological and Otological Society, Inc., will hold its Annual Meeting at the Sheraton-Mt. Royal, Montreal, Canada, May 15-16-17 (mornings only).

Please make early plans to attend the 1956 Spring Meetings in Canada. Both the Seignior Club and Montreal present most attractive features for you and your family. More information about the places will be given later.

Reservations at the Sheraton-Mt. Royal Hotel should be made early by addressing the Reservation Supervisor, 1455 Peel Street, Montreal, P. Q., Canada.

WASHINGTON UNIVERSITY, SAINT LOUIS, DEPARTMENT OF OTOLARYNGOLOGY.

Offers a Basic Science Course in Otolaryngology to start Monday, September 17, 1956. Complete information about the course may be obtained by writing to Theo. E. Walsh, M.D., Head of the Department of Otolaryngology, 640 S. Kingshighway, St. Louis 10, Missouri.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The Sixth International Congress of Otolaryngology will take place in Washington, D. C., from Sunday, May 5, through Friday, May 10, 1957, under the presidency of Arthur W. Proetz, M.D.

The selected subjects for the Plenary (Combined) Sessions to be held Monday, Wednesday and Friday mornings will be:

1. Chronic Suppuration of the Temporal Bone.
2. Collagen Disorders of the Respiratory Tract.
3. Papilloma of the Larynx.

Outstanding internationally recognized authorities will open the discussion of each of these subjects.

Two types of communications are invited: 1. Contributions to the discussions of the selected subjects, limited to five minutes. 2. Original papers, limited to 15 minutes. These should be in one of the four official languages: English, French, German, Spanish.

Motion picture films will be shown continuously except during the Plenary Sessions. There will be both scientific and technical exhibits. Those wishing to submit contributions to the program should communicate with the General Secretary.

Announcement of the Congress has been sent to all otolaryngologists whose names and addresses could be obtained. Additional details concerning registration, housing, entertainment, etc., will be sent to those who have indicated to the General Secretary that they wish further information.

The subscription for Members (physicians) is \$25.00 U.S.A. This includes the privilege of attendance at all official Congress meetings except the banquet for which an additional charge will be made. Other persons accompanying Members may be registered as Associates at a fee of \$10.00 U.S.A.

An interesting program of social functions, visits to points of interest in and around Washington and post-Congress tours is being arranged. The American Express Company is the official travel agent for the Congress. Their offices through-

out the world are available for travel arrangements to the Congress and for post-Congress tours.

All communications should be addressed to the General Secretary, Paul H. Holinger, M.D., 700 N. Michigan Ave., Chicago, Ill., U.S.A.

INDIANA UNIVERSITY MEDICAL CENTER.

The Department of Otolaryngology, Indiana University School of Medicine, offers its annual Anatomical and Clinical Course in Otolaryngology March 26th to April 7th, 1956.

Applicants should address The Post-Graduate Office, Indiana University Medical Center, Indianapolis 7, Indiana.

MOUNT SINAI HOSPITAL

Mount Sinai Hospital offers three post graduate courses given in affiliation with Columbia University. The course on Indirect Laryngoscopy is scheduled for Feb. 13-14, 1956; a course on Voice and Speech Therapy will be given Feb. 15-16, 1956 and the course in Audiology Feb. 17-18, 1956.

These courses may be taken separately or as one combined course.

For further information address The Registrar for Post Graduate Medical Instruction, Mount Sinai Hospital, Fifth Avenue and One-Hundredth Street, New York (29), N. Y.

A special course in Endaural Otologic Surgery will be given by Northwestern University February 28 through March 26, 1956. Course is limited to eight otolaryngologists. Instruction to include lectures, cadaver dissection, etc. For further information write Dr. Geo. E. Shambaugh, Jr., Dept. of Otolaryn., Northwestern Medical School, 303 East Chicago Ave., Chicago (11), Ill.

POSTGRADUATE COURSE.

The Mount Sinai Hospital, New York, in affiliation with Columbia University, announces an intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty, given by Irving B. Goldman, M.D., and staff, July 14-18, 1956.

Candidates should apply to Registrar for Postgraduate Medical Instruction, Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, New York.

UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE.

The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois, College of Medicine, is scheduled for the period March 5 through March 17, 1956. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W. Polk Street, Chicago 12, Illinois.

POSTGRADUATE COURSE IN OTOLARYNGOLOGY.

The Department of Postgraduate Medicine of the University of Michigan Medical School announces the Otolaryngology Conference to be given at the University Hospital, Ann Arbor, Michigan, on April 19, 20 and 21, 1956, under the direction of Dr. A. C. Furstenberg, Chairman of the Department of Otolaryngology at the University of Michigan Medical School.

Guest lecturers of national prominence, together with members of the staff of the Department of Otolaryngology, will participate in the program.

For further information, write to Dr. John M. Sheldon, Director, Department of Postgraduate Medicine, University Hospital, Ann Arbor, Michigan.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Vice-President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Seignior Club, Ottawa, Canada, May, 1956.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Bernard J. McMahon, 8230 Forsyth Blvd., Clayton 24, Mo.
First Vice-President: Robert L. Goodale, 330 Dartmouth St., Boston, Mass.
Second Vice-President: Paul H. Holinger, 700 North Michigan Ave., Chicago 11, Ill.
Secretary: Harry P. Schenck, 326 South 19th St., Philadelphia 3, Pa.
Treasurer: Fred W. Nixon, 1027 Rose Building, Cleveland, Ohio.
Librarian, Historian and Editor: Edwin N. Broyles, 1100 North Charles St., Baltimore, Md.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, Iowa City, Iowa.
President-Elect: Dr. Percy Ireland, Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOTOLOGY AND RHINOLOGY.

Chairman: John R. Lindsay, M.D., Chicago, Ill.
Vice-Chairman: James W. McLaurin, M.D., Baton Rouge, La.
Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Daniel S. Cuning, 115 East 65th St., New York 21, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Sheraton Mount Royal Hotel, Montreal, Canada, May 15-16, 1956 (afternoons only).

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October, 1956.

THE AMERICAN RHINOLOGIC SOCIETY

President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. James Chesson, 1829 High St., Denver, Colo.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Illinois,
October, 1956.
Annual Meeting: Palmer House, Chicago, Illinois, October, 1956.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. D. M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo
2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1956.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pitts-
burgh, Pa.
Secretary: Dr. Louis Joel Felt, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Phila-
delphia 40, Pa., U. S. A.
General Secretary: Dr. C. E. Mañoz MacCormick, P. O. Box 9111, San-
turce 29, Puerto Rico.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and
Broncho-Esophagology.
Time and Place: April 8-12, 1956, San Juan, Puerto Rico.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Hollinger, 700 No. Michigan Ave., Chicago
(11), Ill.
Meeting: Statler Hotel, Washington, D. C., May 5-10, 1957.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. William J. Hitschler.
Vice-President: Dr. Chevalier L. Jackson.
Treasurer: Dr. John J. O'Keefe.
Secretary: Dr. Joseph P. Atkins.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Thomas F. Furlong, Jr., Dr. William A. Lell,
Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, ex-officio.

BALTIMORE NOSE AND THROAT SOCIETY

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Secretary-Treasurer: Dr. Theodore A. Schwartz.

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Vice-President: Dr. Jack Allan Weiss, 109 No. Wabash Ave., Chicago 3, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave.,
Chicago, Ill.
Meeting: First Monday of each Month, October through May.

OTOSCLEROSIS STUDY GROUP.

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Secretary: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1956.

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Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Vice-Chairman: Dr. Edward W. Griffee, 217 Hermann Professional Building, Houston 5, Texas.
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Meeting:

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Vice-President: Dr. Edgar Childrey, Jr., Professional Building, Richmond, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.
Annual Meeting: May 26 - June 2, 1956.

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Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

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Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.

Meeting: George Vanderbilt Hotel, Asheville, N. C., Sept. 16-19, 1956.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

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Meeting jointly with the North Carolina Eye, Ear, Nose and Throat Society, George Vanderbilt Hotel, Asheville, N. Car., Sept. 17-18-19, 1956.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

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LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Secretary of Section on Ophthalmology: Stephen J. Popovich, M.D.

Chairman of Section on Otolaryngology: Herschel H. Burston, M.D.

Secretary of Section on Otolaryngology: Ross A. Goodcell, M.D.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.

Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Lester T. Jones, M.D., 624 Medical Arts Bldg., Portland (5), Oregon.
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Meeting: April 15-19, 1956.

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Meeting: June 6-7, 1956, Chateau Frontenac, Quebec, Canada.

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Meeting:

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